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Blister Rust Resistant Western White Pine for the Inland Empire:

The Story of the First 25 Years of the Research and Development Program

Richard T. Bingham

THE AUTHOR

RICHARD T. BINGHAM, internationally known in the field of forest genetics, retired from the USDA Forest Service in 1974. For 25 years he headed the blister rust resistant western white pine R&D program at the Intermountain Forest and Range Experiment Station's Forestry Sciences Laboratory in Moscow, Idaho. He has authored or coauthored about 25 publications on tree rust resistance genetics. Bingham received his M.S. from the University of Idaho in 1942 in forest pathology. Since retiring, the author, as a Forest Service volunteer, has authored this paper and a guide to Hells Canyon plants. Currently, he is working on a guide to Seven Devils Mountains plants.

RESEARCH SUMMARY

Twenty five years of research and development work (1950-75)—first-phase work undertaken by Forest Service cooperators—has led to experimental production (and soon mass production) of the Inland Empire western white pines bred for blister rust resistance. Breeding has gone through two generations, until 65 percent of the trees resist intense, artificial exposure to the rust fungus. And unless the racial structure of the rust alters disastrously, the long-range survival of these second generation stocks under natural exposure to the rust probably will exceed 65 percent.

Resistance in the second generation stocks is based on selections for general combining ability for a combination of differential and uniform types of resistance. Some of the resistance reactions—and, presumably resistance genes—are identical to those that probably have persisted for long periods near the Asiatic white pine:blister rust gene center. Thus, resistance in these first-phase stocks will probably persist until scientists can produce faster growing and better adapted second-phase stocks embodying more types of resistance genes and more stable resistance.

PROLOGUE

In 1950 four U.S. Department of Agriculture units, which were concerned with the management of western white pine (*Pinus monticola* Dougl.) and control of the white pine blister rust disease (causal pathogen *Cronartium ribicola* J.C. Fisch. ex Rabenh.) in the Inland Empire,[1] began a 25-year program investigating and utilizing genetically controlled resistance to that disease. This research and development venture had a single and practical goal: the rapid and economical development of western white pine planting stock sufficiently resistant and otherwise adapted for Inland Empire planting.

The four USDA units included the Spokane Office of Blister Rust Control (now defunct) of the Bureau of Entomology and Plant Quarantine, and three Forest Service units—the Northern Region, the Northern Rocky Mountain (now part of the Intermountain) Forest and Range Experiment Station, and the California (now Pacific Southwest) Forest and Range Experiment Station. This cooperative work by the four agencies soon became known as the "first-phase resistance program." This implied that any resistant planting stock developed first should satisfy a somewhat limited goal, merely of helping to return western white pine as a manageable component of Inland Empire forests. But also implied in the term "first-phase" was the idea that planting stocks would continue to be improved in subsequent programs—toward successively faster growing, better adapted, and more resistant stocks embodying more resistance genes and more stable resistance.

Twenty-five years of R&D work has seen the first-phase program move through two generations of selection for resistance, culminating with the establishment of seed orchards in 1971-74. By about 1985, the 40 acres (16 ha) of seed orchards should be starting to mass produce second-generation (F_2) seed and 65$^+$ percent resistant F_2 planting stock sufficient for annual reforestation of 10,000 to 20,000 acres (4 050 to 8 100 ha) of prime white pine lands.

Objectives of this paper are three:

1. To assemble under one cover the often obscurely published record of research information already available or produced by this and other blister rust resistance research and development programs.
2. To explain how this body of information was interpreted and used in various blister rust resistance programs.
3. To interpret, as objectively as possible, the extent to which success of our first-phase R&D program was influenced by essentially uncontrollable biological conditions, as well as by extraneous administrative conditions that held at the outset or during the progress of this program.

The approach, not altogether in the order indicated above, is to examine the administrative, research, and biological "climates" in force at the inception of our R&D program; then to explain just how much of our success may have been due to timing of the research work, to the state of research knowledge, to forceful execution of the work, and to administrative or biological happenstances.

The format is one hinging about the 1950 beginning of the program. This format emphasizes conditions and factors that influenced success or failure of the program from its start, and that continued to dog its progress through 25 years to its conclusion in 1974.

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[1]As used here the Inland Empire is meant to be that geopolitical entity including northern Idaho, northeastern Washington, and northwestern Montana; usually the term is extended to include interior south-central British Columbia.

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INTRODUCTION

Some Historical Perspectives

Few forest scientists may aspire to be among those making highly significant contributions to the basic science of plant disease resistance genetics, or even to the art of plant disease resistance breeding. For trees, indeed, make poor materials for basic studies of disease resistance, and research and technology toward developing of disease resistant forest trees will always lag behind the contemporary stage of such work in the annual crop plants. For many years, in fact, tree reproductive and tree disease resistance testing cycles were considered so prohibitively lengthy and costly as to preclude all but a small amount of research. But a substantial and comfortable backlog of basic information on disease resistance has accumulated from the crop plant research (Vavilov 1951). And this legacy has been of great benefit to yesterday's and today's researchers and breeders of disease resistant trees.

State of the Art in 1950

In 1950 it was fair to characterize tree disease resistance genetics and breeding as in its infancy, but as also poised on a threshold of solid research and technology based on work on annual crop plants. Resistance research on three diseases was relatively far advanced because of the epiphytotic nature of the white pine blister rust disease, the chestnut blight (pathogen *Endothia parasitica* [Murr.] P. & H. Anders.), and the Dutch elm vascular disease (*Ceratocystis ulmi* [Buism.] C. Moreau) where introduced among susceptible host populations in Europe and North America.

It is enlightening to look back at early tree disease resistance research and to assess its utility in terms of today's tree breeding programs. Experimentation had begun in the 1910's, but by 1950, aside from a few mostly vegetatively propagated and often horticultural varieties, the widespread use of disease resistant forest trees was almost nil. Nevertheless, a few significant contributions had begun to accumulate. These indicated the progress and promise of tree disease resistance and breeding work in general as follows:

1. Zederbauer (1912) conducted what for the time were quite sophisticated experiments into the development of wind-pollinated, individual-tree progenies of eastern Austrian, western Czechoslovakian, Norwegian, Finnish, and Scottish provenances of Scotch pine (*Pinus sylvestris* L.). These experiments clearly demonstrated intraspecific, less clearly interracial variations in percent of seedlings killed by the endemic needle cast disease caused by *Lophodermium pinastri* (Fr.) Chev. Mortality ranged between 4 and 64 percent in progenies from 12 eastern Austrian and 1 southwestern Czechoslovakian trees, while none of the seedlings from 9 other eastern Austrian and 3 Norwegian, Scottish, or Finnish trees were killed.

2. Van Fleet (1914, 1920) and later Graves (1925) isolated interspecies variations in resistance to the chestnut blight disease caused by *E. parasitica*. The Asiatic chestnuts *Castanea mollissima* Bl. and *C. crenata* Sieb. + Zucc. proved to be quite resistant in respect to the susceptible American chestnut *C. dentata* (Marsh.) Borkh. This work led to many years of interspecies hybridization work, seeking to transfer resistance from the Asiatic chestnuts to the better timber type American chestnut.

3. Moir (1920, 1924) and Spaulding (1922, 1923, 1925, 1929) assembled a massive body of widespread and detailed observations on the relative susceptibility to blister rust of some 16 Eurasian and North American white pines. A very clear picture emerged of the generally greater resistance of the Eurasian species. Interspecies hybridization work, in part seeking to transfer resistance of the Eurasian to the North American white pines, got under way in the late 1930's (Duffield and Stockwell 1949; Johnson 1939a; Righter 1945). About the same time Snell (1931), Riker and Kouba (1940a, 1940b), and Johnson and Heimburger (cf. Farrar 1947) commenced investigating intraspecies variations within *Pinus strobus* L. Before long Riker and others (1943a, 1943b) and Johnson and Heimburger (cf. Farrar 1947) had established that intraspecies variation in blister rust resistance was, in fact, under genetic control, although there were conflicting results as to the degree of its inheritance in wind-pollinated progenies from presumably resistant parental selections.

4. Liese (1930a, 1930b), in experiments with various provenances of *P. sylvestris*, demonstrated interracial variations in resistance to the disease caused by the endemic and autoecious rust *Peridermium pini* (Pers.) Lev. Later, in what for the times were elegant and classic experiments using *P. sylvestris* progenies produced by controlled pollination, Liese (1936) demonstrated that intraspecific variation in resistance to the rust probably also existed within *P. sylvestris*. Small (10- to 23-tree) progenies from the crosses S (susceptible mother tree) × S, S × R (resistant mother tree), R × S, and R × R were found to contain resistant offspring in the ratio 31:52:82:83 respectively.

5. Buisman (1935), sought to replace certain widely used Netherlands elm clones that had proved to be universally susceptible to the introduced Dutch elm disease caused by *C. ulmi*. She demonstrated and isolated intervarietal and interspecific variation in resistance to that disease. This laid the groundwork for today's fruitful Dutch elm disease resistance programs.

From 1920 to 1950 three review articles were published that considered the attainments and promises in the field of breeding disease resistant trees (Hartley 1927; Graves 1948; Clapper and Miller 1949). While these reviews offered a good deal of encouragement as to the likelihood for making genetic gains in forest tree disease resistance, they presented almost no specific data bearing on the genetics and inheritance of resistance. Hartley's (1927) article, however, deserves special mention. His suggestions as to how breeders might use intraspecies variation in resistance to blister rust in *P. strobus* were so perceptive and far ahead of the times as to be almost prophetic (see section on "The Intraspecies Approach").

White Pine Blister Rust Resistance in 1950

Probably the state of the art of breeding for blister rust resistance in white pines in 1950 can best be characterized as "in a state of readiness."

The pathogen *C. ribicola* was quite well understood. We were conversant with its biology and etiology (Klebahn 1905; Colley 1918; Clinton and McCormick 1919). We were particularly aware of the disease's awesome epiphytology as it had been introduced into and spread within planted stands of *P. strobus* in Europe (von Tubeuf 1905–1936; Moir 1920; Spaulding 1922), as well as into native northeastern North American stands of *P. strobus* (Snell 1928; Hirt 1936) and northwestern North American stands of *P. monticola* (Mielke 1943; Buchanan 1938; Buckland 1946) and *Pinus lambertiana* Dougl. (sugar pine) (Mielke 1938).

The technology for resistance testing was largely in hand. Successful methods for inoculating white pine test materials with *C. ribicola* had become available even before 1920 (Spaulding 1912; Clinton and McCormick 1919) and constantly had been improved thereafter (York and Snell 1922; Snell and Rathbun-Gravatt 1925; Hirt 1939; Van Vloten 1939; Riker and others 1943a, 1943b; Slipp 1949). And Cumming and Righter (1948) had just completed an impressive job of improving techniques for controlling pollination in *Pinus*. This meant that reliable methods were available for producing either interspecific or intraspecific hybrid test progenies of guaranteed pedigree.

Two probable sources of genetically controlled resistance to the white pine blister rust disease had been ascertained. There was strong evidence that the several Eurasian white pine species constituted a repository of resistance (Spaulding 1929; Hirt 1940; Childs and Bedwell 1948). And there were persistent reports that rare but presumably resistant individuals were surviving in otherwise severely rust-damaged stands of the susceptible North American white pines (Snell 1931; Schreiner 1938; Riker and Kouba 1940a, 1940b; Farrar 1947; Hirt 1948 in *P. strobus*; Lachmund 1934; Mielke 1943 and Buckland 1946 in *P. monticola*; Mielke 1943 and Childs and Bedwell 1948 in *P. lambertiana*).

Thus, for workers seeking to improve resistance in these commercially important North American white pines, there was a question as to whether it was more efficacious: (1) to introduce the possibly more broadly based and stable resistance from Eurasian white pines that had long been associated with the rust, plus any adaptational problems, via interspecies hybridization; or (2) to seek and utilize intraspecific variations for resistance that appeared to be available in the locally well-adapted, rust-free survivors residual in long and heavily infected stands of native white pines.

Almost nothing was known, except inferentially, about the genetic control of white pine blister rust resistance, and little more was known about the degree of inheritance of resistance as it might pertain to the practical and economical production of resistant planting stocks. In fact, the weightiest evidence obtained in the more critical early studies in Wisconsin and Canada (Riker and others 1943a, 1943b; and Farrar 1947) was contradictory and open to differing interpretations (see section on "The Intraspecies Approach").

Thus, resistance research in the white pines:*C. ribicola* couplet, as that in the *Castanea:E. parasitica* and *Ulmus:C. ulmi* couplets, was poised on a threshold of research and technology from studies on disease resistance in annual crops as well as in trees. All required was a gentle nudge to cause it to fall ahead.

Administrative, Research, and Biological Climates in 1950

The administrative climate in the four cooperating USDA agencies of the late 1940's and early 1950's certainly was favorable. Control of the white pine blister rust disease in the commercial and highly valuable western white pine stands of the Inland Empire had by then become the primary concern of local disease control and forest management personnel (Davis and Moss 1940; Davis 1942; Matthews and Hutchison 1948).

The rust disease had entered several northern Idaho *P. monticola* stands in 1923, but remained undetected there until 1927 (Mielke 1943). Even before the rust was discovered in Idaho, experimental work started toward adapting and improving techniques for eradication of the heteroecious blister rust's alternate hosts (*Ribes* spp., hopefully eradicated to the extent that their populations would be nearly eliminated in and near white pine stands).

By the early 1940's, it was becoming apparent that *Ribes* spp. eradication to a large extent was controlling spread of the rust in *P. strobus* stands of the Northeastern and Lake States, but that it might be far less effective in controlling rust spread between and within Inland Empire *P. monticola* stands (see Ketcham and others 1968 for reasons). In 1937, only 15 years after the rust's entry into Idaho, blister rust disease surveys showed that the average level of infection on young *P. monticola* trees over the entire St. Joe National Forest already had reached 15 percent (Swanson 1939). By the mid–1940's in certain high-rust-hazard areas, infection had climbed to over 95 percent, prospective damage to dominant and codominant white pine poles (crop trees) to over 70 percent—all in less than 20 years (Bingham 1947). As foresters would say, white pine blister rust epiphytotics in Inland Empire western white pine stands were, bar none, the world's most spectacular.

Thus, by 1950, the long and often discouraging blister rust control battle in the Inland Empire was almost 25 years old. Most key control personnel had been on the job, except as interrupted by World War II, for 15 to 25 years. Those administrators tenacious enough to stay with the fight were a strongly motivated, highly experienced, and close-knit group with exceptional morale. They learned the hard way to be adaptable and receptive to the implementation of newer and possibly better means of control.

The research climate of the early 1950's was almost, if not quite, as favorable. Not until the mid-1950's and early 1960's did the forestry research funds and work force begin to multiply. Research funds had lagged about 5 years behind compared to the postwar buildup in rust control. Thus, in 1950, ample blister rust control funds were available for undertaking developmental work on resistance, but research funds were not. However, since there was the usual grey area as to what constituted development and what research, there was scant criticism when financially adroit control administrators saw fit to subsidize some of the preliminary research.

Perhaps most important, by 1950 the materials for researching blister rust resistance (here rust-free parent selections gleaned from rust-decimated *P. monticola* stands) had, biologically, reached a particularly advantageous stage in their development. Our unpublished data reveal that at that time our blister rust epiphytotics could be characterized by susceptible:healthy tree ratios of from more than 375 dead and dying crop trees:1 surviving crop tree, or even up to 10,000 ± infected trees:1 completely rust-free tree. Obviously, under these conditions strong selection pressures had been exerted on the existing generation of white pines by the rampant rust disease. Remarkably rust-free trees remained, and after more than 25 years of annual assault by the rust, it was difficult to accept the hypothesis that they were merely "escapes." Conversely, it was easy to accept the hypothesis that the strong selection pressure exerted by the rust had exposed most of the susceptible individuals, or to assume that healthy survivors of the epiphytotics were indeed genetically resistant.

Thus, the early 1950's seemed almost ideal for starting a program of researching and using heritable blister rust resistance in Inland Empire *P. monticola*. The overall climate hardly could have been better—administratively, financially, or biologically. All that was needed for success was a moderately competent research and development team. If team members had the ability to adapt existing research findings and technology to their needs, and if they had a little more biological good luck, success would be theirs.

REVIEW OF THE PRE-1950 LITERATURE

The Interspecies Approach

Seeking to transfer inherent resistance from Eurasian white pine species.—The useful concept of "gene centers" was not available in 1950 (see Leppik [1970] on the gene center of the white pines:*C. ribicola* couplet). Nevertheless, much evidence had accumulated on the existence of geographic areas with repositories of tree disease resistance: in the *Castanea:E.* parasitica couplet (Gravatt and Gill 1930; Gravatt 1949; Graves 1950); the *Ulmus:C. ulmi*:couplet (Buisman 1935); and the white pines:*C. ribicola* couplet. A question remained as to whether there was a single Asiatic center from which *C. ribicola* had spread to Europe and America (Spaulding 1929; Leppik 1970), or if there were two such centers, one in Asia and one in the European Alps (Gäumann 1948).

Very early Tranzschel (1895) had noted the differential susceptibility (or resistance) of the Asiatic white pine *Pinus sibirica* DuTour and the European white pine *Pinus cembra* L. Tubeuf's observations (1917, 1920, 1926, 1933) then further clarified the situation in the white pines:*C. ribicola* couplet by emphasizing the high susceptibility of most North American white pines (particularly *P. strobus*) when compared with Eurasian white pines. Pennington and others (1921) extended these findings on the relatively high susceptibility of the North American white pines (*P. strobus* and *Pinus flexilis* James) in relation to the European *P. cembra* in small Rhode Island plantings.

Later Moir (1920 and 1924) and Spaulding (1922, 1923, 1925), as official U.S. Department of Agriculture observers reviewing the status of white pine blister rust disease in Europe, brought back hundreds of detailed observations on the relative susceptibility of seven Eurasian white pines and nine North American white pines where widely planted in western Europe and Scandinavia. Their keen and widespread European and North American observations on the blister rust epiphytotics then developing in *P. strobus* and *P. monticola* stands, led to Spaulding's preliminary (1925) and final (1929) classifications of the relative blister rust resistance of some 16 of the world's 20-odd white pine species. Spaulding (1925) attributed part of the resistance of the Eurasian white pines to the relatively short number of years they retained their needles, and possibly also because their stomata tended to be restricted only to the outer surfaces of the needles.

These early observations pointed out the need for clearer delineation and quantification of this interspecies variation in resistance. Ultimately they led to the establishment of more sensitive tests made under North American conditions including one within the range of *P. strobus* in central New York (Hirt 1940), the other within the range of *P. monticola* in southern British Columbia and near Mt. Hood in northwestern Oregon (Childs and Bedwell 1948). In table 1, salient results of these two tests are tabulated and compared with Spaulding's (1929) observations.

The practical implications of these findings slowly became apparent. The immediate prospect was that piecemeal introduction of resistant Eurasian species might fill some of the gaps left by European or North American blister rust epiphytotics. However, on closer inspection, it soon appeared that silvicultural limitations (slow growth, procumbent habit, rapidly tapering bole form, coarse branching, and cold sensitivity) would preclude introduction of most Eurasian species and provenances for other than experimental purposes.

More troublesome, it also became apparent that it was difficult, if not impossible, to secure any reasonably useful amounts of seed of the faster growing, better formed, and cold-hardy provenances of the more promising resistant species *P. peuce, P. griffithii, P. armandii*, and *P. koraiensis*. This was because these species remained little known and quite inaccessible (see p. 97-271 in Bingham and others [1972] for detailed information on the distribution and intrinsic qualities of the world's white pine species). Any direct and extensive introduction of resistant Eurasian white pine thus never really got under way.

A second prospect (and the main subject of this section) was that the resistance of the Eurasian white pine might be transferred to the very susceptible but nearly ideal timber type, North American white pines (*P. strobus, P. monticola* and *P.*

Table 1.—Review of pre-1950 investigations into the relative blister rust resistance of various white pine species

Taxonomic section subsection species[1]	Common name	Main distribution	Spaulding (1929)		Hirt (1940)		Childs and Bedwell (1948)		
			General classification	Rank[3]	Numerical classification	Rank[3,5,6]	Classifications		
							General	Numerical[7]	Rank[3]
STROBUS									
STROBI									
Pinus armandii Franchet	Armand pine	Central and Southwestern China	Immune	1[4]			Resistant	1.0	1
Pinus griffithii McClelland	Blue pine	Northern Pakistan and India, Nepal and Bhutan	Resistant	3			Resistant	2.0	2
Pinus peuce Grisebach	Balkan pine	Yugoslovia, Albania, and Bulgaria	Resistant	4	0.39	4	Moderately susceptible	1.7	5
Pinus parviflora Sieb. & Zucc.	Japanese white pine	Japan	Resistant	8[4]					
Pinus strobus Linnaeus	Eastern white pine	Eastern United States and Canada	Susceptible	10	.71	6	Susceptible	3.6	7
Pinus ayacahuite Ehrenberg	Mexican white pine	Southern Mexico	Susceptible	11[4]					
Pinus lambertiana Douglas	Sugar pine	Southern Oregon and North-central California	Susceptible	12[4]			Exceedingly susceptible	6.2	10
Pinus strobiformis Engelmann	Southwestern white pine	Southwestern United States and Northern Mexico	Susceptible	13[4]	.69	5	Susceptible	3.0	6
Pinus flexilis James	Limber pine	Southwestern Canada and Central Northwestern Interior United States	Very susceptible	15	1.01	7	Susceptible	3.8	8
Pinus monticola Douglas	Western white pine	Southwestern Canada and Northwestern United States	Very susceptible	16	1.16	8	Very susceptible	4.7	9
CEMBRAE									
Pinus cembra Linnaeus	European stone pine	European Alps	Resistant	2	.00	1	—	—	—
Pinus koraiensis Sieb. & Zucc.	Korean pine	North Korea and Eastern China	Resistant	7[4]	.11	3	Resistant	2.0	3
Pinus sibirica DuTour	Siberian stone pine	Siberia and Northern Mongolia	Susceptible	9			—	—	—
Pinus albicaulis Engelmann	Whitebark pine	Southwestern Canada and Interior Northwestern United States	Very susceptible	14			Most susceptible	—	—[8]
PARRYA									
BALFOURIANAE									
Pinus aristata Engelmann[2]	Bristlecone pine	Interior West-Central United States	Resistant	5[4]	.03	2	Moderately susceptible	1.3	4
Pinus balfouriana Grev. & Balf.	Foxtail pine	Central and Northern California	Resistant	6[4]				—	—[9]

[1]Taxonomy follows Little and Critchfield (1969).
[2]Recently P. aristata has been separated into two species (Bailey 1970).
[3]Rankings are in order of increasing susceptibility.
[4]Spaulding's (1929) rankings were based upon observations of small numbers of trees.
[5]Hirt's (1940) numerical classification was based on $\sqrt{\text{no. cankers/M}}$ needles inoculated, expressed as the number of cankers greater (+) or lesser(-) than found on *P. strobus* (in other words, *P. strobus* = 0.00). Here, Hirt's $\sqrt{\text{no. cankers}}$ are rearranged to show their variation between the most resistant species (*P. cembra* = 0.00 and the most susceptible species (*P. monticola* = 1.16) tested.
[6]The rankings are the author's, but they follow Hirt's (1940) numerical rankings.
[7]Childs and Bedwell's (1948) numerical ranking was based on number of cankers produced from one million needles exposed to *C. ribicola* for one season. Here the author has averaged these numbers from Childs and Bedwell's table 1, across one to seven different tests.
[8]*Pinus albicaulis* was not included in Childs and Bedwell's (1948) main field tests, probably because it became so severely infected in the nurseries before transplanting to the field test plots. However, in two other studies (Bedwell and Childs 1943), it supported so many more cankers than *P. monticola* (6.4 to 20 times more cankers) that they ranked it as even more susceptible than *P. lambertiana*.
[9]*Pinus balfouriana* had such low nursery survival that it was not entered in Childs and Bedwell's (1948) main field tests. However, they found it "often infected" in an arboretum near Carson, Wash.

lambertiana), via interspecies hybridization. This prospect was especially attractive for a number of reasons:

1. Biologically, such hybrids indeed were feasible. At least three different spontaneous hybrids involving the better Eurasian and the North American white pine species (from within subsection Strobi) already had been reported as occurring naturally in European or North American arboreta and gardens (one each by Jackson 1933, Rehder 1940, Sax 1947).
2. Remarkable success already had been achieved in transferring disease resistance by interspecies and intervarietal hybridization in annual crop plants (tobacco, cotton, potatoes, and others), and then by restoring quality and yield through repeated backcrossing to the commercial variety (Thomas 1952). Promising early results already had been obtained in transferring chestnut blight resistance from the resistant Asiatic chestnuts *C. mollissima* and *C. crenata* to the better timber type but highly susceptible American chestnut *C. dentata* (Clapper and Gravatt 1936; Graves 1940).
3. Certain hybrids, notably in maize after outcrossing of repeatedly inbred lines (see Jones 1920 and East 1936), were exhibiting a strikingly increased yield, and this "hybrid vigor" seemed to hold, at least for the juvenile *P. strobus* × *P. monticola* F_1 hybrid (Righter 1945; Buchholz 1945; Stockwell and Righter 1949).

As a result, a flurry of new work began in the production and testing of white pine species hybrids. At first reports concerned mainly the biological feasibility, production technology, and potential hybrid vigor of the F_1 hybrids (Duffield and Stockwell 1949; Righter 1946; Johnson and Heimburger 1946; Righter and Duffield 1951). Soon Righter (1946) outlined the possibilities and economics of mass production of pine hybrids. Through 1950, Righter and Duffield (1951) had reported successful, artificial production of 10 different white pine hybrids, eight of them involving resistant Eurasian white pines.

From this early work in interspecies hybridization, it emerged that: (1) crosses between resistant Eurasian species and susceptible but commercially important North American species within taxonomic subsection Strobi were mostly successful, except those involving *P. armandii* or *P. lambertiana*, which mostly were unsuccessful or difficult; and (2) intersubsectional crosses involving resistant Eurasian stone pines of subsection Cembrae and the susceptible North American species of subsection Strobi were mostly unsuccessful or difficult (see table 1 for taxonomic subsections of white pine species).

Results of nursery or field tests of these hybrids to determine their blister rust resistance, or their long-range adaptation and growth, were still awaited in 1950.

The Interracial Approach

Seeking to use any provenance-related resistance in *P. strobus*.—In retrospect, it is curious that in European experiments of the late 1930's a definite attempt was made to isolate interracial variations for blister rust resistance among *P. strobus* provenances. Although today we might predict only a low, probably chance possibility for the existence of such racial variation, in the light of the times its existence would have seemed quite reasonable. The literature of the period, in fact, was liberally sprinkled with reports of racial variations in tree disease resistance—in the *Pinus sylvestris:Peridermium pini* couplet (Liese 1930a, 1930b), the *Pseudotsuga menziesii* (Mirb.) Franco:*Rhabdocline pseudotsuga* Syd. couplet (Rohde 1934), and others.

This search for interracial resistance may have started from a suggestion of Liese (1936). Based on his finding of provenance-related resistance in the endemic and more or less balanced *Pinus sylvestris:Peridermium pini* couplet (Liese 1930a, 1930b), he suggested the extension of that finding to the epiphytotic and unbalanced *P. strobus:C. ribicola* couplet.[2] Liese's (1936) suggestion came from Liro's (1907) assumption that the existence of racial variation for blister rust resistance in *P. strobus* long since had been demonstrated by Eriksson (1896) and by Tranzschel (1895).

Eriksson's (1896) evidence, however, really was flimsy. He observed what he considered to be wide variation in susceptibility to blister rust in two nursery beds that contained 7- to 8-year-old *P. strobus* plants each from a different seed source. However, one bed contained only 10 plants. And Tranzschel (1895) actually observed what he considered to be varietal, not interracial, resistance to blister rust demonstrated in adjacent seedbeds of the then-defined Siberian and Alpen varieties of *Pinus cembra*. Today these varieties are considered quite discrete and geographically well-separated species, *Pinus sibirica* and *P. cembra* (Little and Critchfield 1969).

Van Vloten (1939, 1941) instituted a fairly large Dutch experiment, exposing seedlings from various provenances of *P. strobus* to *C. ribicola* spreading from interplanted *Ribes nigrum* L. bushes. Initially Van Vloten (1941) noted rather uniformly heavy infection in all provenances as disclosed by typical needle and bark lesions, but also that formation of aecia varied widely (between 0 and 72 percent). Later Van Vloten (personal communication of Feb. 14, 1956) reported that 98 to 100 percent of the plants in all provenances had become infected and that most of them died on transplanting.

This sort of research was also under consideration in Germany. Heimburger (1956) reported that in 1937 he had been engaged in collecting *P. strobus* seed for proposed German experiments on racial variations in blister rust resistance.

The Intraspecies Approach

Seeking to use any resistance available in rare rust-free selections of North American white pines.—As far as can be determined, Dr. Carl Hartley (1927) of the U.S. Department of Agriculture, Bureau of Plant Industry, Division of Forest Pathology, was the first scientist to advocate intraspecies breeding (within the species *P. strobus*) to isolate and utilize genetic resistance to the white pine blister rust disease. Hartley apparently based some far-reaching recommendations on resistance breeding on the performance of a single *P. strobus* tree that Dr. John Shaw Boyce at the Yale University School of Forestry had observed to be tolerating attack by *C. ribicola*. Nevertheless, Hartley's prediction on the latent nature of resistance in North American white pine, and his several suggestions as to how researchers might explore and use intraspecific variation in *P. strobus*, were so remarkable for the time that they are worth repeating in more modern terminology:

1. Given time, natural selection favoring resistance would uncover latent resistance first in *P. strobus*, later in *P. monticola*, by directing attention to those individuals that survived the ongoing blister rust epiphytotics. Also, the general level of resistance in *P. strobus* probably would be higher than that so far found in *Castanea dentata* to the chestnut blight.

[2]This and all subsequent footnote material found in the text (not on tables) is anecdotal. Thus, these footnotes are removed from the text to the appendix.

2. "Line selection" for resistance could be started immediately in *P. strobus*. This artificial selection would be applied merely through collections of wind-pollinated seed from the widely dispersed survivors that might be found early in the wake of still-advancing blister rust epiphytotics.

3. In the future, "crude mass selection" would be applied by the rust itself, in time leaving small, yet intercrossing populations of surviving, resistant *P. strobus* trees, from which such mass-selected seed might be collected.

4. One "less empirical procedure" for resistance breeding should be followed. This involved checking the resistance of a number of epiphytotic-surviving and presumably resistant parent selections by exposing 50 or more of their vegetative propagules (grafts), alongside ordinary nursery or control seedlings, for 10 to 15 years in a disease garden with planted *Ribes* spp. bushes. Finally, the disease garden would be converted into a clonal seed orchard by culling the susceptible clones and controls.

5. Another "less empirical procedure" would be to establish the disease garden with offspring from controlled crosses among the presumably resistant parent selections. This would determine which parents transmitted the highest levels of resistance to their seedling progenies. The researcher could then establish clonal seed orchards with grafts taken from those parental selections that transmitted the highest levels of resistance. This procedure was apparently the suggestion of Hartley's colleague, Dr. Wilber Brotherton, Jr., a crop plant disease specialist also with the Bureau of Plant Industry.

The events that followed showed just how perceptive were Hartley's (1927) predictions as to the latency and nature of blister rust resistance in North American white pines, and just how farsighted were his suggestions as to profitable breeding methods. In fact, each of his suggestions is in use today in one or another of the present programs for blister rust resistance breeding.

Thus, quite soon and precisely in line with Hartley's (1927) prediction, remarkably blister-rust-free and probably resistant survivors began to appear, with increasing frequency, in the progressively more and more heavily rusted stands of North American white pines. They appeared first in *P. strobus* stands (Snell 1931; Schreiner 1938; Riker and Kouba 1940a, 1940b; Farrar 1947; Hirt 1948), later in similar *P. monticola* stands (Lachmund 1934; Mielke 1943; Buckland 1946) and *P. lambertiana* stands (Mielke 1943; Childs and Bedwell 1948).

Next, using Hartley's (1927) suggestion on "line selection," Snell (1931) tested exposure of wind-pollinated progeny from a single, presumably resistant New Hampshire *P. strobus* selection to heavy, natural inoculation with *C. ribicola*. The result, in Snell's words as later quoted by Mirov (1938), was: "The trees from the resistant pine were not more resistant than trees from normal seed." Snell qualified this by remarking, "Of course, it must be held in mind that no one has any information regarding the staminate parent in the production of these seeds, but it is extremely likely that the pollen entering into combination came from susceptible parents for the most part." Later Snell (personal communication of March 30, 1973) noted that by 1934—5 years after first rust exposure—the seedlings from the New Hampshire selection were 58 percent infected, while control seedlings were 48 percent infected.

Before long a much larger and more conclusive test of this "line selection," as well as of Hartley's (1927) suggestion for confirming resistance of parental selections via their vegetative propagules, was being set up in the Lake States. These new tests were part of a pioneering, blister rust resistance R&D program led by the University of Wisconsin (Dr. A. J. Riker, plant pathologist), in cooperation with the USDA Bureau of Entomology & Plant Quarantine (T. F. Kouba and L. E. Byam, blister rust control officers), and the Wisconsin Conservation Department (W. H. Brener, nurseryman).

First, working in natural, Wisconsin *P. strobus* stands they considered to be heavily blister rusted, the cooperators selected individual, rust free white pines and collected scionwood and wind-pollinated seed therefrom (Riker and Kouba, 1940a, 1940b). Next, they grafted scions from the resistant parental selections on ordinary *P. strobus* rootstocks, pruned away rust susceptible rootstock foliage, and exposed the grafts along with wind-pollinated seedlings of the same selections to natural and artificial blister rust inoculation in a blister rust disease garden near Wisconsin Rapids, Wisc. Soon (as did Snell, via Mirov, 1938 and via Bingham, personal communication) the Wisconsin cooperators were reporting discouraging results in respect to the transmission of resistance to wind-pollinated offspring of rust free selections. At the same time, however, they reported that grafts from most of the same selections appeared to be resistant (Riker and others, 1943a, 1943b).

The initial test (Riker and others 1943a, 1943b) included wind-pollinated progenies from 63 Wisconsin *P. strobus* selections, exposed in the disease garden in two naturally inoculated and two naturally and artificially inoculated replicates. Each replicate included both wind-pollinated progenies and grafts from the same "resistant" parent selections, plus ordinary and presumably susceptible *P. strobus* control seedlings. In the case of the wind-pollinated seedlings from selections, results showed that an average of 78 percent of 384 control seedlings, versus 75 percent of the 1,494 seedlings from resistant selections, supported blister rust stem cankers only 12 months after inoculation. These results came from averaging the percentages of infections for the two artificially inoculated replicates.

In identical test replicates the Wisconsin researchers produced some encouraging results on performance of vegetative propagules from the same 63 *P. strobus* selections. By averaging their results from the two artificially inoculated replicates (as done above) it appeared that only 9 percent of 156 grafts from resistant selections were stem cankered 12 months after inoculation. Also, there were outward and visible evidences of genetically controlled resistance and resistance mechanisms on grafts (Riker and others 1943b, 1949, 1953). These included small size of foliar lesions (under 1/2 × 1 mm), apparent failure of rust mycelium to extend from infected needles into the branch or stem bark, bark lesions that were reduced both in number and size, bark lesions that were "corked out" and with rust fungus therein presumably dead, relatively slow extension of bark lesions (in branches, to the extent that the fungus failed to reach the stem only a few inches away before the branch died), and failure of branch and stem lesions to produce aecia.

Based on these preliminary results, Riker and others (1943a, 1943b, 1949) drew the following inferences:

1. Continued survival of parental selections, overall high resistance of their grafts, and the outward signs of resistance and resistance mechanisms thereon all indicate genetic control of blister rust resistance in *P. strobus*.

2. Overall low resistance of wind-pollinated progenies from parental selections indicate that any resistance the offspring had inherited was being masked, either by (a) dominant susceptibility-gene(s) of the unknown but probably predominantly susceptible pollen parents, or (b) the overwhelming severity of the natural and artificial inoculations of the tests.

3. Emphasis should be placed on vegetative propagation and toward reproducing those parental selections whose grafts resist infection (an inference already drawn from Snell's data by Mirov [1938]).

Exercising traditional "good hindsight," it seems all three of these inferences should have been drawn only with certain reservations. The first was questionable because "graft-controls" (grafts from ordinary susceptible trees, preferably of the same ages and localities as the resistant selections) were not included in the tests; therefore, any physiological effects of graftage on resistance could not be appraised. Much later Patton and Riker (1958b) and Patton (1961) showed the pronounced effect of parent tree age on apparent resistance of grafts.

The second inference also was open to question because: (1) lack of control of pollination perhaps should have precluded the making of any working assumptions as to the gene-control that was being exercised in the inheritance of resistance, and (2) if there existed some lower "field level" of resistance, then why was this threshold resistance not overcome in the grafts as well as in the wind-pollinated seedlings?

The third inference as to the desirability of emphasizing work on vegetative propagations of *P. strobus* also was open to question. It assumes that the first two somewhat shaky inferences were acceptable. It also apparently was based on what might prove to be overly optimistic predictions on significant seed production on young grafts and on the acceptance of somewhat shaky financial risks.

For instance, it was predicted that grafts from 30-year-old resistant selections could be planted where mostly isolated from outside pollination and induced to produce cones and seed (at least adequate for further testing) within as little as 4 to 5 years. This time frame seems unduly optimistic for white pines even in view of promising results then commonly being reported for other conifers (Lindquist 1948; Syrach Larsen 1956). The shaky financial risk lay in the possibly low resistance level that might result in the "control-pollinated" progenies obtained by wind pollination within the isolated graft planting. Testing for resistance (say over 10 years—5 years for graft seed production and 5 years for resistance testing) might prove that inherent levels of resistance were too low for practical planting use. If so, then any use of the experimental graft planting as a seed orchard might have to be abandoned, and the planting perhaps used only for further testing with the polycrossed "control-pollinated" progenies.

In any event, these early inferences or preliminary conclusions of Riker and others (1943a, 1943b) persisted in the literature for more than 10 years. They trickled into the review literature (Graves 1948; Clapper and Miller 1949), and may have delayed not only the Wisconsin but other programs (Buckland 1946) for developing blister rust resistant planting stocks. Seven years later Riker and others (in a paper presented in Stockholm in 1950, but delayed in publication until 1953) first reported that wind-pollinated seedlings in progenies from certain "most resistant" parental *P. strobus* selections indeed had survived the heavy inoculation of their tests in "significantly better" proportions than had control seedlings. Just how much "better" had been survival in these particular, open-pollinated progenies was not specified.

In the Wisconsin program, perhaps unfortunately, a good deal of emphasis was shifted to research into vegetative propagation of *P. strobus* and to the establishment of graft outplantings throughout northern Wisconsin (Patten and Riker 1966). Cuttings taken from older trees, such as the 25- to 40-year-old Wisconsin selections, rooted only from 1 to 20 percent in extensive trials (Thomas and Riker 1950). Thus, the technology for economical mass-production of vegetative propagules of older *P. strobus* selections simply defied development.

Meanwhile, Canadian workers, apparently from experiments planned and installed about the same time as the Wisconsin experiments, were coming up with quite different and much less clear results. According to Farrar (1947), Johnson and Heimburger's tests, made under the auspices of the Canadian National Research Council and the Dominion Forest Service, included some "40 different strains." Presumably these included wind-pollinated stand collections and wind-pollinated progenies from individual "resistant" *P. strobus* selections. The tests were made mainly in a Montreal disease garden and included unidentified University of Wisconsin and Harvard University strains, along with wind-pollinated progenies coming from noteworthy survivors found in a rust-decimated, Pointe Platon, Quebec, plantation. Farrar (1947) reported that after 8 years of exposure to natural inoculation by *C. ribicola* in the disease garden, certain unidentified strains of *P. strobus* became almost 100 percent infected while others remained almost free of the rust. Unfortunately, the Canadian program was interrupted when researchers transferred to other work and other stations; in fact, Heimburger (1956) was later unable to find, reassemble, and reassess the original data.

A pertinent question remains about the contradictory nature of the early Wisconsin and Canadian results. Why was it that Brotherton's suggestion (Hartley 1927) concerning the probable utility of control-pollinated test progenies was never pursued? Today it seems obvious that by controlling pollination, the researchers would produce much sounder evidence concerning the nature and inheritance of any resistance-genes; also, that findings would become available in one short (say 5- to 7-year) pollination and resistance test cycle. They would also answer important questions about the level of resistance to be obtained in the first breeding generations and about whether that level was adequate for using such "early generation" stocks for practical reforestation.

One answer might be that foresters and forest pathologists of the time (the author included) had only sketchy training in genetics, or even hesitated to extend to trees the successful research and technology from agronomic work. Or perhaps the researchers had not been exposed to the new technology for controlling pollination in pines. This technology, although dating at least from the early 1930's (Liese [1936] had reported the use of cloth bags to control pollination in *P. sylvestris*), had not been refined and made generally available until the late 1940's (Cumming and Righter 1948).

In respect to physiologic or pathogenic races of *C. ribicola*, almost nothing was known through 1950. Hahn (1949a, 1949b) had discussed the continuing immunity of certain red and black

currant varieties. He suggested that as long as existing and new races (if any) all gave the same reaction on these varieties, then, new races capable of attacking the immune currants probably had not appeared. Riker and others (1943b) and Boyce (1948) merely mentioned that there were then no evidences for *C. ribicola* races.

Summary of Past Research

Any new programs for developing intraspecies variation in resistance were the beneficiaries of considerable research information that would pave the way for new and probably profitable research. It appeared that blister rust resistance was under genetic control. Resistance already had been isolated by natural selection in Eurasian white pines, and rapidly was being isolated and made available through the application of strong selection pressures in force in heavily rusted stands of North American white pines. Also, while genetic control of resistance appeared not to be as simple as could be hoped, and while important questions remained as to the genetic control, mechanisms, and practical utility of resistance, it seemed that these problems might be resolved by following quite uncomplicated and economical investigative pathways.

The Forest Service's first-phase R&D program for improving blister rust resistance in Inland Empire western white pine benefited greatly from this past research and problem analysis. Indeed, given financing, a reasonably well-qualified research team, continued enthusiastic support from potential consumers, and a little more biological good luck, the program seemed almost bound to succeed.

BEGINNINGS OF THE FIRST-PHASE PROGRAM

Blister Rust Situation by the 1940's

From 1946 to 1950 there remained a large, war-delayed backlog of blister rust control, *Ribes* spp., eradication work in the Inland Empire. The awesome spread of the disease in stands of the susceptible *P. monticola* had accelerated, especially in 1937 and 1941, when the weather was highly favorable to spreading rust (Paine and Slipp 1947). By the late 1940's, infection and damage in all ages of *P. monticola* stands was becoming highly visible throughout the region. Many stands already contained high proportions of rust-killed and multicankered trees. Thus, control work was assuming a more desperate urgency, soon to be reflected in increasing appropriations for Federal and State control or in increasing control assessments levied against the privately owned white pine lands (under State law by the northern Idaho Timber Protection Associations). But while control appropriations were increasing, those for tree disease research (including blister rust research) remained pegged at relatively low wartime levels.

This lopsided situation developed for several reasons. Foremost, a vocal and effective coalition of interested citizens and private white pine landowners was pressing for increased control appropriations, if not for research. White pine stocking and blister rust damage surveys showed that, while rust damage often was severe, because of excess stocking adequate numbers of white pines survived to stock most *P. monticola* stands and to justify control work. And the newly developed herbicides 2-4-D and 2-4-5-T were at hand, promising much more effective and economical eradication of *Ribes* spp. Administation of disease research, however, was well aside from this mainstream of control activity. It was isolated mainly in another Federal bureau (Plant Industry, Soils, and Agriculture Engineering) and division (Forest Pathology) where increased funding was proving to be much harder to come by. Small, field-service units, staffed with forest pathologists from the division, were widely scattered around the West (Portland, Berkeley, Albuquerque, Logan, Fort Collins). These units were already overextended on regional forest pathology problems. They found scant support for taking on any new research related to the white pine blister rust disease. Meanwhile, indirect but strong support was coming from the Northern Rocky Mountain (now Intermountain) Forest and Range Experiment Station whose timber management researchers were acutely aware of the blister rust problem in *P. monticola* and of the research hiatus.

In response to this research vacuum, the Federal agency in charge of blister rust control on western State and private white pine lands—the Division of Plant Disease Control of the Bureau of Entomology and Plant Quarantine (BEPQ-PDC)—organized a western Development and Improvement (D&I) Unit headquartered at Berkeley, Calif. This unit was assigned to develop and improve methods of blister rust control. For the Inland Empire these responsibilities were delegated to a three-man D&I subunit stationed in Spokane with the BEPQ-PDC Office of Blister Rust Control. The subunit also serviced the needs of the Forest Service Region 1 Division of Timber Management. This division worked in six "white pine" National Forests in undertaking blister rust control.

This bureaucratic maze developed largely because of the way various blister rust control and research funds were appropriated on both the State and Federal levels. It was perpetuated because of reorganizational delays inevitable under a rapidly developing blister rust control scene. How this nightmare of a field control organization operated at all is a mystery. But operate it did, under the guidance of some experienced and dedicated administrators. It persisted through 1953 when a national reorganization placed all the Inland Empire control work under a Forest Service Region 1 Division of Blister Rust Control, and all Inland Empire disease research under the Forest Service Northern Rocky Mountain Forest and Range Experiment Station.

Facts and Fancies on the Planning

In 1946, the author was the junior member of the newly established, three-man Spokane D&I subunit. I was stationed in Spokane at the BEPQ-PDC Office of Blister Rust Control, working out of a small laboratory on the eighth floor of the downtown Realty Building. As a forest pathologist, my responsibilities included development of blister damage survey methodology.

From time to time during survey work, I came across rare *P. monticola* individuals that somehow, although growing in heavily blister-rusted stands, had remained free of the rust. Found in 1946, the first of these remarkable trees was full-crowned, dominant, 60 years old, and almost 100 ft (30 m) tall. It was the only white pine crop tree among almost 380, sampled across a heavily rusted 530-acre (215 ha) stand, that was

completely free of either living or dead blister rust cankers (fig. 1). This tree was the more remarkable to me because I had shared in the vicarious pleasures of climbing, examining, and discarding several other such "resistant" selections because of many, hidden but active cankers—much to the chagrin of the hopeful, but earthbound, supervisory personnel below.

Through the next 3 years, 14 more such rust-free individuals were located in five other heavily infected Inland Empire, *P. monticola* stands. Following procedures on selection and testing of *P. strobus* used in Wisconsin, I commenced small cutting-rooting trials in 1949. These trials were housed in a sort of greenhouse that protruded precariously about 4 ft (1 + m) from an eighth floor window of the Spokane lab. Also, with Cumming and Righter's new (1948) publication on controlling pollination in *Pinus* at hand, I climbed selection No. 1, the 100-ft (30-m) tree described above. There, I attempted a controlled self-pollination. Not only did the cuttings fail to root, but the self-pollination failed to produce any filled seeds. However, these abortive trials may have served other much more useful purposes.[3]

Figure 1.—Resistant *P. monticola* selection No. 1; neighboring white pines were multi-cankered or killed by blister rust.

The lack of forest genetics and tree improvement expertise was recognized at once, and the cooperators requested help from another Forest Service unit—the California (now Pacific Southwest) Forest and Range Experiment Station's Institute of Forest Genetics.

The Office of Blister Rust Control, cognizant of the lack of research funds, labeled the new resistance work as "developmental," and requested that the D&I subunit assign the author up to one-third time on the new work. The Northern Rocky Mountain Station also assigned A. E. Squillace, research forester, up to one-fourth time on the work. The Division of Timber Management of Region 1 came up with $5,000 in National Forest blister rust control funds to finance a five-man search for additional rust-free selections. And Dr. F. I. Righter, director of the Placerville, Calif., Institute of Forest Genetics of the California Station, agreed to train Bingham and Squillace in the methodology of controlled pollination and tree improvement. Righter also assigned Forest Geneticist Dr. J. W. Duffield to the training and planning work.

The Placerville training session, held in 1950, constituted the first real meeting of the hastily assembled investigative team of Bingham, Squillace, and Duffield. It was fortuitous that these men were both professionally and personally compatible. We immediately set out to define our problem and the research required for its solution. In those days, research planning procedures were flexible, and much of the problem analysis and study planning work proceeded informally largely by unwritten cooperative agreement. The research team did feel obliged to produce one semiformal document—a magnificent, huge flow chart covering the what, when, and who of interlocking research jobs. The problem analysis drew heavily on the early results of Snell, Riker and coworkers, and of Johnson and Heimburger, as well as on the lucid and farsighted suggestions of Dr. Carl Hartley (1927). You may be sure that the points enumerated in the following section were not nearly so precisely or logically defined or stated. Time and the first few years of field work have polished them.

A Skeleton Problem Analysis and Study Plan

WORKING ASSUMPTIONS

1. Inland Empire *P. monticola* stands included some of the world's most spectacular white pine blister rust epiphytotics. Heavily rusted stands of highly susceptible *P. monticola* had by 1950 undergone 20 to 25 years of exposure to the virulent disease. These stands did, however, contain surviving and completely rust-free individuals. These individuals appeared to be highly resistant phenotypes that were isolated by strong natural selection pressure generated by the epiphytotic rust disease. The "resistant" selections seemed to be just as good (and in the long run proved to be far better) as those already isolated and tested in the *P. strobus* programs.
2. There were two research jobs of the highest priority—verifying genetic control of resistance in *P. monticola*, and determining whether the level of inheritance of resistance in early generation offspring from potentially resistant phenotypes would justify a practical breeding program.
3. Mass vegetative propagation of any resistant *P. monticola* clones would be impractical, neither technologically nor economically attainable. Vegetative propagation would be used only as a research tool or as a means for establishment of clonal seed orchards.

[3]See appendix for anecdote.

4. Aside from the overwhelming problem of susceptibility of existing stands to *C. ribicola, P. monticola* came close to being an ideal, timber-type conifer. It had proved to be eminently manageable; it reproduced naturally and easily, grew relatively rapidly, and competed well in either managed or unmanaged mixed stands (Haig 1932; Haig and others 1941). The wood was light-colored, soft, straight-grained, and easily sanded, glued, or painted. As finish sash and trim lumber, the wood had continued to command the region's highest stumpage and lumber prices (Betts 1940; Matthews and Hutchison 1948). Furthermore, it was more than acceptable as an addition to the region's mixed-species paper pulp output (personal communications, local pulp mills). Thus, it seemed appropriate to confine the major emphasis toward improvement of blister rust resistance of *P. monticola* to the intraspecies breeding option, working mostly within that valuable and locally well adapted species. A secondary emphasis would be the introduction of germ plasm from resistant Eurasian white pines through interspecies hybridization. It appeared that such interspecies work was already well under way at the Placerville Institute of Forest Genetics (Righter 1945; Duffield and Stockwell 1949), at Harvard University (Sax 1947), and in Canada (Johnson 1939a, 1939b; Johnson and Heimburger 1946).

5. Level of inheritance of seed-transmitted resistance had remained obscure and conflicting during the testing of wind-pollinated seedling progenies of resistant *P. strobus* phenotypes—for instance, the results of Riker and others (1943a, 1943b, 1949) vs. those of Johnson and Heimburger as reported by Farrar (1947). Therefore, it would be advantageous to certify male parentage by controlling pollination and restricting it to crosses or selfs among phenotypically resistant *P. monticola* selections. Although we planners at the time didn't know, this same assumption apparently had already been reached by Riker and his coworkers in Wisconsin (Patton and Riker 1958a).

6. To avoid unnecessary delay, controlled-pollination work would be undertaken immediately, directly on the phenotypically resistant *P. monticola* selections in the forest.

7. The difficulty and high cost of this work, and the consequently high value of control-pollinated seed (later estimated at $5,000 to $10,000 per pound or $11,000 to $22,000 per kilogram) would necessitate the development of surefire methods for seed pretreatment to secure good germination and thereafter for minimizing losses of seed and nursery test seedlings.

8. Many more phenotypically resistant selections should be located for undertaking the preliminary and necessary resistance research. As it turned out, we found a total of 58 selections by the time pollination work began in June 1950.

SELECTION CRITERIA

1. Artificial selection work should be directed only toward resistant phenotypes that were reproductively mature.

2. Artificial selection, to be superimposed on the hypothesized fabric of preexisting natural selection, should seek to emphasize the effects of that natural selection. Per-generation genetic gain in resistance should be maximized by restricting selection to those areas and stands that had undergone the stiffest natural selection pressure; this also would minimize the inclusion of selections that were merely chance "escapes" from the disease. For practical purposes, this came down to undertaking artificial selection work only in stands that had undergone more than 20 years exposure to the rust, and where severity of that exposure was attested by multiple cankerings of the average living or rust-killed white pine. It also came down to restricting selection to completely rust-free individuals chosen on the basis of a close, branch-by-branch, internode-by-internode, top-to-bottom examination, for both living and dead cankers.

3. Tandem, index, or other forms of multiple-trait selection toward improving growth rate, branching habit, planting adaptation, and so forth, along with rust resistance might be possible if a sufficiently large nucleus of resistant selections would transmit useful levels of resistance to their seed-propagated offspring. Thus, while overriding importance should be on blister rust resistance, such features as growth rate, or branching habit should be assessed, and the inheritance of these traits followed to the extent possible to gain breeding information from resistance or supplementary progeny tests.

TESTING CRITERIA

1. A reliable assessment of resistance should be obtained in complete, randomized-block progeny tests, if the number of blocks were large enough to iron out effects of unequal rust exposure and other extraneous variables. Requisite numbers of blocks or of test seedlings within blocks were unknown. But for a start, 9 blocks containing 10-seedling family row plots would be used. Later this was upped to 10 blocks and 16-seedling plots but with only moderate gains in experimental sensitivity.

2. To shorten test-rotation years and to minimize requisite numbers of test blocks by increasing uniformity of rust exposure, all progeny tests would be artificially inoculated. Because earlier research had defined the sensitivity of young white pines to the blister rust disease, particularly 1-year-old plants (Clinton and McCormick 1919; York and Snell 1922), probably only 2-year and older test progenies should be inoculated. Assistance would be needed in establishing a satisfactory and repeatable methodology for large-scale artificial inoculations, and this would be sought from Division of Forest Pathology blister rust specialists.

3. The outward appearance and the timing of the various symptoms of the blister rust disease or signs of the blister rust fungus was quite well established on young *P. monticola* plants (Lachmund 1933; Kimmey 1940; Slipp 1949), and routine symptom-sign inspections could be timed and carried out with some confidence. But the outward appearances of any resistance reactions, or the best timing of inspections for them still was ill defined (Riker and others 1943b, 1949). Thus, a more or less continuous, sample inspection schedule would have to be maintained, both to elucidate any new or different resistance reactions and to time inspections for the determination of their significance.

At the close of the Placerville session in early April 1950, two more jobs remained to be done before the program could get under way during the anticipated June pollination season. The first job was to increase the number of rust-free and reproductively mature selections so that an adequate first-year crossing program might be undertaken. As already mentioned, the Region 1 Division of Timber Management had earmarked $5,000 of their blister rust control funds for this job, and the money went to hiring a temporary, five-man, tree-search crew for May and June 1950. This crew, supervised by the author, searched close to 1,000 acres (400 ha) of white pine stands in seven heavily infected areas near places where the blister rust was introduced in 1923–27. By late June, the crew had increased rust-free selections from 14 to 58.

The second job was to purchase control-pollination supplies and to assemble various pollination syringes, pollen extractors, and so forth. For this first, short-notice season, the Institute of Forest Genetics loaned us 500 of their standard, heavy-canvas, acetate-film-windowed pollination bags, as well as serially numbered airplane cloth pollination streamer tags. The rest of the gear was assembled by the research team.

The First Controlled Pollination Season, 1950

About June 15 the D&I team of Squillace, Duffield, and Bingham reassembled at Blister Rust Control (field) Headquarters, Clarkia, Idaho, where we were near most of the 58 rust-free selections. Tony Squillace came from Missoula bringing the godsend of a ramshackle, prewar, carryall vehicle. And Jack Duffield drove from California in D&I boss Harold Offord's car lugging a bulky load of pollination bags and tags from the Institute of Forest Genetics.

Clarkia BRC Headquarters was an age-mellowed, and already long defunct, railroad-logging base camp. It had seemingly numberless barns, sheds, bunkhouses, and warehouses, plus a rectangular "roundhouse" with 15-ft tall (5-m) doors for servicing long-gone logging locomotives and rolling stock. Altogether there was just about one-half acre (0.2 ha) of roofed space where a single winter snow removal required a week's work by a 5- to 10-man crew. The old camp housed an assortment of carpentry, plumbing, vehicle repair, machine, canvas fabrication and repair shops, and other buildings. Such facilities were necessary for supplying up to 1,000 control workers scattered over the St. Joe National Forest area in as many as 50 separate, road or mule-pack tent camps. It also housed a wonderfully supportive group of BRC "overhead" personnel who, through the years, fed (oh, so well) and housed (only one bedbug outbreak) the researchers and helped them in the evenings with everything from assembling pollen extractors to extracting and cleaning seeds. The entire layout was rented for $25 a month from a latter-day and benevolent landlord, Potlatch Forests, Inc. (now Potlatch Corp.); and we were shocked and incensed when, about 1955, PFI doubled the monthly rent in order to meet taxes.

None of us researchers will forget that first and hectic pollination season. It taxed the considerable physical abilities of the young, three-man team near their limits. There were no summer field assistants, but, fortunately, neither were there prescribed limits like 8-hour days or 40-hour weeks.

The team was completely inexperienced in controlled pollination of *P. monticola* under northern Idaho conditions. We installed only about 600 pollination bags and worked in only about 25 trees that had produced male or female strobili—all within 15 miles (25 km) of our headquarters. We found later that with experience we could put in the same hours and amount of work to handle two to three times that number of trees and pollination bags. Three visits to each tree top were minimal the first season—once to detect and bag female strobili and collect pollen, once to pollinate and tag bagged strobili, and once to remove pollinaton bags and check success of pollinations. But with more fruitful trees that bore 100 to 400 female strobili, we found ourselves climbing them 20 or more times.

Three of the worst scenarios we could think of all came true: First, we climbed the tree so prematurely that we were unable to differentiate female strobili from vegetative buds, or that we overlooked the very small and green clusters of male strobili. Second, we bagged the female strobili so early that on rapidly elongating, current season shoots we had to slip the bags out on the shoots one or more times lest shoots become curled up inside the bags. And third, we bagged weak and usually single female strobili on the tree crown's inner and lower branches that most often "pooped-out" (aborted), but not until they reached "buds open" stage and were pollinated repeatedly, only to drop off when we removed the pollination bags.

Indeed, we literally wore out some of these more fruitful trees climbing them so many times. The soft bark on the upper sides of branches in the upper third of the tree crowns was completely crushed and destroyed even by our sponge rubber-soled boots. These trees had to be "rested" for a year or two thereafter.

By the late 1950's Squillace and Bingham, with three or four field assistants, were undertaking 4,000-bag pollination seasons involving over 100 trees scattered along 400 miles (640 km) of backwoods roads, and with relative ease.

Our main problem emerged when we attempted to follow the quite sophisticated methods of Cumming and Righter (1948) for extracting uncontaminated pollens. These methods that worked so well in warm and dry California were beset with serious problems in the relatively cold and damp *P. monticola* pollination season in the northern Idaho woods. When we followed the recommended procedure of washing clusters of male strobili and entering them under water into sterile canvas-topped, metal-funnel-bottomed pollen extractors, it was only the very ripest or often already-pollen-shedding clusters of male strobili that could be induced to shed pollens. These pollens were mostly very damp and soon molded. Prebreakfast and postdinner were heralded by cacophony from us three scientists on the bunkhouse porch when we vigorously rapped with wooden sticks on the metal extractor funnels hoping to shake down enough fresh, if damp, pollen for a day's work. Even the innovative Duffield's pollen extractor air manifold, designed and produced almost overnight in the plumbing and carpentry shop, failed to dry out the stubbornly sodden mass of pollen catkins—and this after a full night of blowing woodstove-heated bunkhouse air up through the bottom of the extractor funnels.

Fortunately, the pollen extractions almost daily provided viable, if damp, pollens that proved to be capable of effecting fertilization. Cones were collected September 6 to 19, 1951, just as they commenced opening. They were spread out by individual crosses in deep wooden, window-screen bottomed drying trays, and racked up ceiling-high alongside the bunkhouse barrel stove. After the author and the volunteer Clarkia BRC overhead crew hand extracted, winnowed, and cleaned the seed[4], the result was as follows: of 93 separate, control-pollinated crosses attempted on 25 different selections, 78 of 83 intraspecies crosses, and 4 of 10 interspecies crosses (all × *P. strobus*), yielded adequate numbers of filled seeds for progeny testing. The gods of fertility had indeed favored us mightily!

Between pollination and cone collection we experienced the usual problems with cone and seed insects, and a few problems with squirrels[5]. Fortunately, Jack Duffield was aware of the insect problem and insisted that we rebag control-pollinated cones their second year; thus, we were on hand to do this job in late April or May, as soon as snow permitted. Even as early as we cone-bagged[6], the cone beetle *Conophthorus ponderosae* Hopkins, and less often two cone moths (*Eucosma rescissoriana* Heinrich and *Dioryctria abietivorella* [Groté]), would beat us to as many as 25 percent of the cones. But sans bagging, cone insect losses were known to have exceeded 90 percent in certain areas and years (Barnes, Bingham, and Schenk 1962).

By 1951, mostly in the course of other work, we researchers had found 12 more rust-free selections in three other areas. This brought the total to 70, which we considered adequate for preliminary investigations.

Controlled pollination work, however, was continued to 1953 when after 4 years we had nearly 200 *P. monticola*, first-generation (F_1), intraspecies progenies in hand or coming by fall 1954. We considered these 200 progenies to be adequate for preliminary investigations into genetic control and level of inheritance of blister rust resistance. After 4 years, it was evident that there were no practical means by which we could produce a complete diallel cross for testing. Successful crossing of each selection with every other selection would have required many more years of pollination work.

Preliminary Progeny Testing, 1952 to 1959

Controlled pollination work for the 4 years 1950 to 1953 had resulted in about 200 F_1 seed progenies from intraspecies crosses among about 40 of the rust-free parent selections. The seed progenies became available in the 4 seed years, 1951 to 1954, and were sown 1952 to 1955 in 4 successive progeny tests in a small nursery in Spokane, Wash. A "1952 progeny test" was established that spring with seeds from the 1950 controlled pollinations; a "1953 progeny test" in that spring with seeds from the 1951 controlled pollinations; and so forth.

Each progeny test contained several lots of presumably susceptible, control materials. In the 1952 progeny test, control lots were ordinary Forest Service Savenac Nursery *P. monticola* seedlots. Thereafter, controls came from mixtures of wind-pollinated seeds from differently colored cones found in selection area squirrel caches, or from mixtures of wind-pollinated seeds from equal numbers of cones from five or six obviously susceptible (multi-cankered) trees in various selection areas. Many wind-pollinated seedlots collected from the rust-free selections were also included in these progeny tests.

The pollination and progeny test operations are outlined in table 2, using the 1952-sown test as an example. Most of these operations are also detailed in the next several pages.

[4]See appendix for anecdote.
[5]See appendix for anecdote.
[6]See appendix for anecdote.

Seed Pretreatment and Germination

It is difficult to secure consistently good and rapid seed germination in most 5-needled white pines, and *P. monticola* is no exception. To satisfy the species' after-ripening requirements and to break seedcoat and other forms of dormancy, the seeds in our progeny tests received more than 90 days of cold-moist, "stratification" treatment at 35° to 40° F. (1.7° to 4.4° C) in the refrigerator (Larsen 1925).

First, in late January, we cold-soaked the seed in 35° to 40° F (1.7° to 4.4° C) tap water overnight, drained the excess water, and dusted the wet seed with Fermate. Next, for the 1952 seed only, we mixed the wet seed with a minimum of fine, sterilized sand and put this mixture in water-permeable, sausage-casing packets salvaged from used pollination bags. Packets varied in sizes depending on the size of the seedlot. Finally, we laid the seed packets on their sides, one layer thick, between l-inch (2.5-cm) layers of premoistened sphagnum peat, with about 10 layers of seed packets to a bottom-drained but lidded, 10-inch (2.5-cm) diameter × 12-inch (30-cm) tall metal freezer can. These cans then were stored in a 35° to 40° F (1.7° to 4.4° C) refrigerator.

Theoretically the low pH "soil solution" should have been diffused from the wet peat layers into the sand-and-seed-filled packets, suppressing fungal and bacterial action. And the seed could readily be separated from the sand merely by flushing away the fine sand through a seed-retaining sieve. Unfortunately, things didn't quite work out this way. Instead, at the close of the stratification period (over 90 days) and just a weekend away from the proposed April sowing, Tony Squillace and I panicked when we found most of the larger seed packets had become soft bricks with the seed and sand cemented by a dense network of tough hyaline fungal mycelium that was firmly attached to the seed coats. Some of these bricks required an hour or more to dismantle, alternately stirring and spraying, and finally handpicking each seed from the tenacious mat of fungal mycelium. Seedcoats were severely eroded, but most endosperms appeared to be firm and the seed looked and smelled all right. Nevertheless, our hearts were in our throats for fear that we might have negated 2 years of work by our failure to detect and control this fungus problem when periodically aerating and checking moisture level of seed packets.

But again we were lucky; following the April 29 sowing, seedling emergence was 85 percent—a level seldom attained in more than 15 years of subsequent tests. Even so, we hastened to change our seed stratification procedures. For the next few years we substituted very fine, dry, screened sphagnum peat for the fine sand inside the seed packets. This controlled fungal and bacterial activity quite effectively, although many of the moist and expanded peat particles would not flush through the sieve when we were recovering seed at the end of the stratification period. We also increased the stratification period to about 120 days. Effective and safe fungicides such as Captan, permitting the relative ease of naked stratification, were still things of the future.

Table 2.—An outline of pollination and progeny testing operations using the 1952 progeny test as an example

Year	Months	Operation	Seedling age	Months after artificial inoculation
			Years	
1950	June-July	Female strobili of rust-free selections isolated in pollination bags; pollens collected and extracted; bagged strobili pollinated and tagged.		
	July-Aug.	Pollination bags removed and success of pollinations estimated.		
1951	May	Cloth cone bags installed over 2d year conelets.		
	Sept.-Oct.	Mature cones collected and separated according to pollination tags into various crosses; seed extracted, cleaned, and counted; progeny test plans made; and corresponding numbers of 60-seedling progeny test nursery flats constructed, filled with forest soil in tarpaper plant bands, tagged, and stored (see fig. 2).		
1952	Jan.	Seed packeted and cold-moist stratified by crosses.	1	
	April	Progeny test established, sowing 1–4 stratified seeds of each test progeny in each of 9 10-plant-band rows in a 9 complete randomized block design (ultimately toward 90 test seedlings per progeny).		
1953	Sept.-Oct.	Test seedlings artificially inoculated under inoculation tents with sporidia shed from telia on heavily infected *Ribes* spp. leaves (almost exclusively 1953, 5-needle-bundle, secondary foliage present); three outplanting plots cleared, fenced, cultivated and planted with *Ribes* spp. bushes.	2	0
1954	May	Seedlings reduced to 1 per plant band, pruning out all but the centermost.		
	June-July	Seedlings transplanted, 3 rows each with 10 seedlings (30 seedlings of 3 randomized blocks) being outplanted onto each of 3 field plots.		
	July-Aug.	Five-needled, 1953 foliage examined for presence of blister rust needle lesion symptoms.		10–11
	Aug.-Oct.	First natural inoculation of 1953 or 1954 foliage, rust spreading from *Ribes* spp. planted on or occurring near outplanting plots.	3	11–13
1955	July-Aug.	Residual 1953 foliage reinspected for missed or latent needle spots from 1953 artificial inoculation, and 1954 foliage for spots from 1954 natural inoculation; seedling 1953 stem and branch internodes examined for rust bark lesions developing from artificial inoculations; abnormal bark lesions that might represent resistance reactions identified and described, and any seedling deaths caused by rust identified.	4	22–23
1956-1957	July-Aug.	Seedling 1953–56 stem and branch internodes examined for rust bark lesions from artificial and natural inoculation; seedlings classified in various living categories, or as rust-killed; progeny test considered to be essentially complete 4 years after artificial inoculation and with rust from 3 years of natural inoculation visible.	5–6	34–47

In another aspect of seed germination, we were decidedly unlucky in the first, or 1952, progeny test. This was in our reliance on Savenac Nursery to provide us with germinable control seedlots. Four of the five seedlots supplied by the nursery germinated at less than 5 percent, and we suddenly found our progeny tests were essentially without controls. Fortunately the nursery at Haugan, Mont., had sown several other germinable *P. monticola* seedlots in fall 1951, and these were available, as cotyledon-stage seedlings, to replace our four nongerminable lots.

We carefully lifted the small seedlings from four Savenac Nursery lots, discarding any with broken radicles. Seedlings were then rushed to Spokane and transplanted into pencil-sized dibble holes flushed full of a soil slurry. The transplanting was better than 90 percent successful, but unfortunately the transplants remained dwarfy and of lower than expected susceptibility for several years thereafter.

Progeny Testing Cycle

Using the 1952, or first-sown, test as a trial, the progeny testing cycle was devised and altered as we went along. By the end of this first test, we were able to set timing and methodology for the three subsequent preliminary progeny tests. The cycle involved 2 years of nursery and transplanting operations (including one artificial inoculation with pine-infecting *C. ribicola* basidiospores). This was followed by 4 or more years of individual seedling inspections (on field outplanting plots) aimed at detecting presence and development of the disease in foliage and bark, as well as any resistance reactions in the seedling host plants. Actually, beginning with controlled pollination for production of test progenies, the test cycle extended over 8 or more years. Methodology used in the nursery and field plot operations is detailed in the next several pages. Throughout the progeny testing, seedlings were routinely weeded, fertilized, and sprinkler irrigated as necessary in the nursery or field plots.

FIRST YEAR, SEED-SOWING

Stratified seed was sown in late April to early May in a prescribed number of 12- by 20-inch (30- by 50-cm) western redcedar flats each 8 inches (20 cm) deep (fig. 2). These were then plunged to ground line, back to back, in a small Spokane nursery (fig. 3). Depending on the amount of seed available and stratified, one to four seeds of each test progeny were centered or evenly distributed across the forest-soil surface and covered with 0.25 inch (0.6 cm) of white sand, in each of 90 heavy, open-ended tarpaper plant bands 2 inches by 2 inches by 8 inches (5 cm by 5 cm by 20 cm)that were soil-filled and arranged randomly as nine 10-band rows scattered through the 60-band flats. This experimental design was devised by Tony Squillace; it included nine complete randomized blocks, each block containing one randomly located, 10-seedling row-plot (the basic test replicate) of each test progeny.

SECOND YEAR, ARTIFICIAL INOCULATION

From mid-September to early October, all test seedlings (2 years old) were artificially inoculated once for 72 hours. This procedure was devised and tested in 1951 and early 1952 by cooperating forest pathologists J. W. Kimmey and C. D. Leaphart, respectively, of the Berkeley, Calif., and Missoula, Mont., field offices of the Division of Forest Pathology. Inoculations took place at the end of the test seedlings' second growing season when the seedlings' foliage was almost exclusively composed of 5-needle, secondary needle bundles.

Inoculation proceeded inside shaded, presoaked, and intermittently fogged canvas inoculation tents. Inoculum was mature but usually ungerminated *C. ribicola* telia borne on the undersides of *Ribes hudsonianum* Richards var. *petiolare* (Dougl.) Jancz. and/or *Ribes viscosissimum* Pursh leaves. The infected leaves were collected on the woody *Ribes* spp. branches, the freshly cut lower ends of which were plunged into the wet nursery soil between the test seedlings (fig. 4).

Telial formation on the infected *Ribes* spp. leaves already had been triggered by falling autumn temperatures in the mountains of northern Idaho and northwestern Montana where we collected the inoculum; and when it was cool but dry, we could collect leaves with ungerminated telia. Thereafter, we tried to maintain low temperatures of 55° to 65° F (13 ° to 18° C) inside the shaded and evaporatively cooled inoculation tents. At the same time we tried to maintain relative humidities

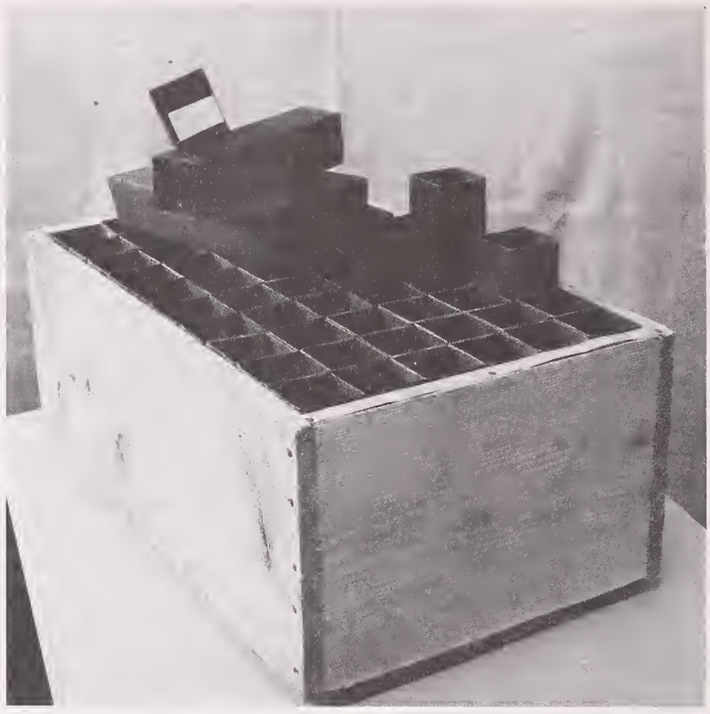


Figure 2.—Western redcedar flat containing sixty 2- by 2- by 8-inch (5- by 5- by 20-cm) tarpaper plant bands, pretagged to show the identities of six different 10-seedling rows of test progenies along the upper leading edge.

Figure 3.—Double rows of cedar flats containing soil-filled plant bands, in the Spokane blister rust nursery, spring 1952. Volunteer helpers Dick Watt (foreground) and Cap Larsen (checkered coat) are sowing *P. monticola* seeds on the surface of each plant band, following a guide card to locate the nine flats and 10-seedling rows therein for the particular test progenies, then covering the seeds in the rows with sand.

Figure 4.—*Ribes* spp. branches with germinating *C. ribicola* telia on undersides of leaves. The branches were stuck in wet soil in and around *P. monticola* test seedlings in two Spokane nursery beds. Central row of fog nozzles over the nursery bed aisle helped maintain high relative humidity inside the inoculation tent.

near the 100 percent level by fogging inside the tents and spraying tents with water from the outside. If these conditions were ideal, after about 18 hours the teliospores of the telial columns would have germinated, basidia and basidiospores would have formed, and the basidiospores would be starting to shed. Shedding of basidiospores would continue for most of the remaining time in the 3 days of inoculation (Hirt 1942).

Basidiospore casts were not estimated in 1953 when the first or 1952-sown test was inoculated, but basidiospore casts were estimated thereafter from 1954 to 1956. Estimates of the cast, as collected on vaseline-coated slides, ranged from 6 to 53 basidiospores per square millimeter, and estimating that the average 2-year-old *P. monticola* seedling presents a 1 000 mm^2 foliar target, then the foliage of the average test seedling could have intercepted 6,000 to 53,000 basidiospores.

Artificial inoculation proved to be the least controllable, and thus the most critical, operation of the entire test cycle (Bingham 1972; Patton 1972). This was mostly because we were dependent upon naturally produced inoculum from the field, or because we were inoculating large numbers of test seedlings outdoors in the nursery. In both places we were at the mercy of the weather. Cool, wet weather for a day or two preceding and during the inoculum collection usually meant that most of the teliospores in *C. ribicola* telial columns had germinated, produced basidia and basidiospores, and already had shed most of their basidiospores. Conversely, hot, dry weather during the 3-day nursery inoculation usually meant that we had off-and-on, nighttime-only basidiospore production and infection of test seedling foliage. This happened because we were unable to maintain the required low temperatures and high relative humidities in the tent-covered nursery beds during all the daytime hours.

THIRD YEAR, OUTPLANTING TEST SEEDLINGS

Already, at the age of 2 years, the test seedlings grown in the Spokane nursery were becoming crowded (fig. 5). We lacked transplanting space there, yet feared possible wildfire losses on any single field outplanting site. So we cleared, cultivated, and fenced three geographically separated outplanting sites in 1953, the year before the first transplanting. These sites were situated in heavy blister rust infection centers, one along Elk Creek about 3 miles (5 km) above Elk River in Idaho, one just across the St. Maries River west of Fernwood, Idaho, and one about 5 miles (8 km) up Randolph Creek northwest of Saltese, Mont. Each of these plot sites also contained a 1951 and 1952 planted test of grafted, clonal lines from 36 of the earliest found rust-free selections (Bingham 1966).

Figure 5.—Two-year-old *P. monticola* seedlings, showing root growth between plant bands. For transplanting, rows of seedlings were separated, lengthwise and crosswise, by slicing with a sharp butcherknife. (Photo courtesy Francois Mergen)

Three of the nine randomized blocks of test seedlings (in other words, 30 of the test seedlings) were outplanted onto each field plot at the start of their third growing season, in June and early July. Transplanting was done by lifting the 60-plant band flats from the Splkane nursery, transporting them to the field site, dismantling the flats, cutting both ways between the rows of plant bands that were by then interconnected by root growth (fig. 4), and finally by sliding the still-tubed seedling into a transplanting hole. These holes had been precut along 9-foot (2.7-m) low row-plot lines, each hole spaced 1 ft (30.5 cm) apart along a board template. The soil plugs, just the size of each tubed seedling, had been cut and removed from the hole using a soil plug cutter (fig. 6) designed by the D&I Unit's mechanical engineer, John F. Breakey.[7] Survival of the seedlings, enhanced by banding and sprinkler irrigation, reached almost 100 percent.

Squillace's experimental design, established in the nursery at 2-inch (5-cm) square seedling spacing was merely expanded six times and carried into the field intact at 1 ft (30.5 cm) square spacing. Each seedling was maintained in the field in the same relative position as in the 10-seedling nursery rows, or in relation to the adjacent nursery rows and seedlings as they occurred in the nursery flats. Flats had been tagged with the randomly drawn test progeny identities (such as female 58 × male 17, 58 × Wind, Control C, and so forth). Once the 50-lb (23-kg) flats had been transported onto the outplanting plots,

[7]See appendix for anecdote.

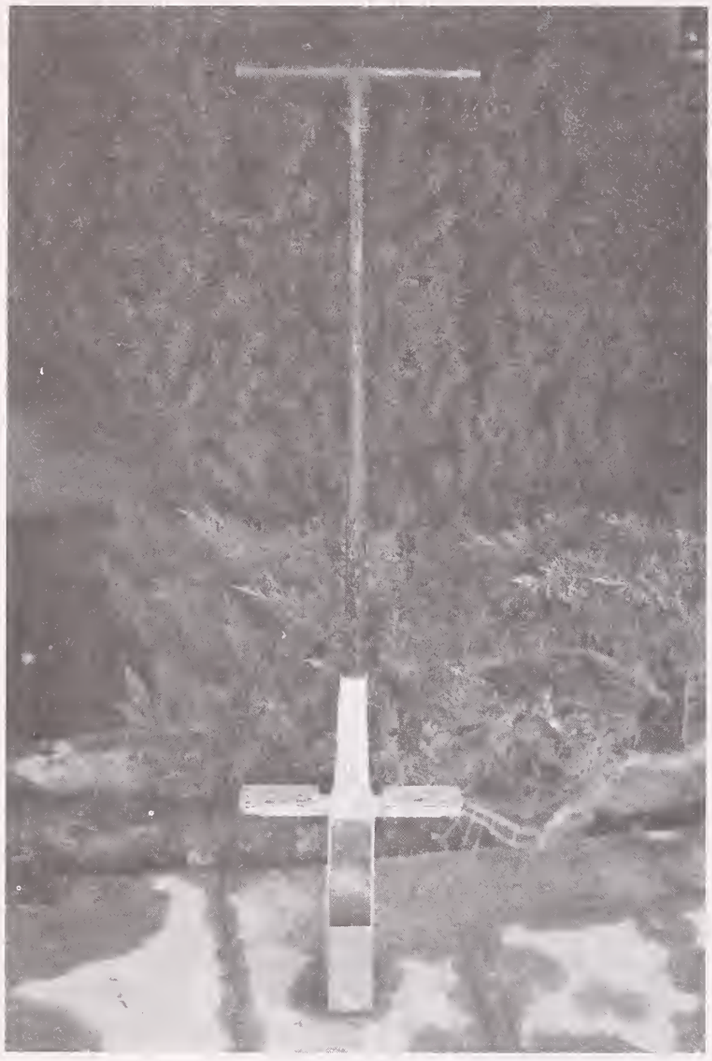


Figure 6.—John Breakey's soil plug cutter. It removed a 2- by 2- by 8-inch (5- by 5- by 20-cm) soil plug for transplanting plant banded *P. monticola* seedlings.

they were placed near the six row-plot stakes that corresponded to the flat and progeny identities. The flats were then opened along one side as shown in figure 5, each successive row-plot of 10 seedlings was removed from the flat, and the individual seedlings were transplanted into the precut holes in sequence of rows and by position of seedlings within the rows. Row-alinement was maintained by extending the 9-ft (2.7-m) plug-cutting template board enough so that both ends lined up with a row-plot stake in the row being planted and the corresponding stake in the next bed over.

To augment artificial inoculation should infection therefrom prove to be light, we had already planted rows of *Ribes viscosissimum* bushes 1 ft (30.5 cm) off either end of the row plots.

This exact spacing of seedlings and progeny row-plots proved to be useful methodology. Twenty-five years later we could identify each progeny and the individual seedlings therein with certainty. And we could map the relative success of artificial inoculations. Lightly or uninfected portions of the seedling beds soon became apparent in the field, and the underexposed seedlings therein could be recognized and eliminated from resistance investigations.

In early July 1954, Tony Squillace and I, along with summer assistants George Blake and Eugene Amman, were transplanting the last third of the 1952 progeny test seedlings onto the relatively high-elevation Randolph Creek, Mont., plot. Patches of snow were still visible on the mountainside above the plot site. Our first job was to hand-carry some 135 flats, each more than 50 lb (23 kg), downhill 50 yards (45 m) from the road, across Randolph Creek on a slippery footlog, and then 150 yards (135 m) up a steep hillside onto the cleared plot site. For the safety of the seedlings, we carried the angular and difficult-to-balance flats against our bellies with the seedlings a scant 12 inches (30 cm) beneath our eyes. From this particular angle, we soon noticed that the foliage of most of the seedlings was liberally sprinkled with a multitude of yellow spots or flecks (fig. 7). This was the first time we had observed this needle-spot phenomenon, and because it offered a good excuse to rest, a hasty, plotside consultation ensued. Sure enough, the needle spots resembled those described and pictured in the literature as the first, visible symptoms of the blister rust disease. We had probably not noted the spots while planting the two low-elevation plots because seedling flats for those plots had been removed from the relatively warm Spokane nursery as much as a month earlier than the Randolph Creek seedling flats.

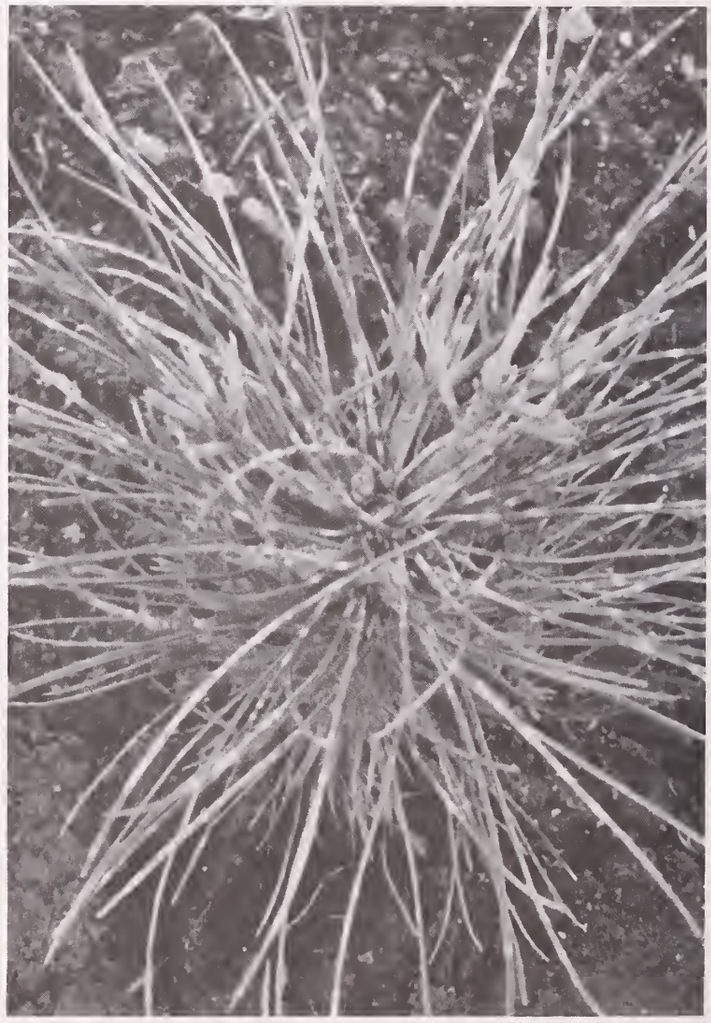


Figure 7.—Multiple blister rust needle lesions on a susceptible *P. monticola* seedling that is starting its third season of growth, about 11 to 12 months after artificial inoculation at Spokane.

Variation in frequency of needle spots seemed to be associated with different test progenies and parents, and, undeniably, we were quite excited. What wonders we had wrought! Here was our first vision of genetic control of resistance, if only we could decide how to measure it! We were mighty tired from the long hours and weeks of transplanting, but the only decision that could be made was to immediately start another round of the field plots to gather needle-spotting data.

THIRD YEAR, INSPECTION FOR FOLIAR INFECTION

We were quite hesitant at first to designate any foliar lesion as a bona fide blister rust symptom. After all, all we had to go on were a few written descriptions and line drawings or black and white photos (Clinton and McCormick 1919; Spaulding 1922; York and others 1927; Pierson and Buchanan 1938). Furthermore there were similar, yellowish-green discolorations associated with various mechanical injuries (such as cracked, broken, or insect-chewed needles). So we made microscopic examinations of frozen thin sections that disclosed rust hyphae and especially the massive rust pseudostromas (fig. 8) that underlay genuine rust foliar lesions. With time and experience, we were soon accurately identifying the quite discrete, circular, lemon to orange-yellow blister rust needle spots.

One measure of the authority of our diagnoses can be obtained from the performance of the presumably nonresistant control seedlings. Here, among seedlings of five different control lots used in the 1952 progeny test in July and August 1954, we diagnosed 235 seedlings as having blister rust foliar lesions on foliage of the 1953 internode. Within 2 years, 95 percent of these infected seedlings had produced one or more typical blister rust cankers in bark of the 1953 internode. Ultimately, 99 percent became cankered on 1953 or later internodes. On the other side of the ledger, among the total of 7,523 controlled and wind-pollinated seedlings of rust-free selections inoculated on 1953 foliage, 6,293 were found to be spotted in 1954 and 1955 examinations. Only 303 more seedlings later developed needle spots or cankers on the 1953 internode. We either missed spots, or there were none on less than 5 percent of the infected trees.

It can be inferred then that in the 1952 progeny test, the single fall 1953 artificial inoculation was quite successful. In fact, of the 7,523 controlled and wind-pollinated seedlings of rust-free selections that were inoculated, 6,293 or 84 percent were designated as needle-spotted 22 to 23 months later. However, we detected variations in the degree and uniformity of artificial inoculation in two respects. First, there was more than 20 percent difference between the foliar infection from a first inoculation run in blocks 1 to 6, and that from a second run in blocks 7 to 9. Second, the rust epidemic maps disclosed that seedlings in certain parts of the nursery beds—notably on outside or end rows—were not as heavily or completely spotted. Fortunately, over time, natural inoculation tended to iron out these differences. But over the years, making improvements in inoculation methods as we could, we never were able to eliminate such sources of variation (as shown by fig. 9, which maps intensity of needle-spotting in the 1964 progeny test).

Another thing that continued to plague us over the years was our difficulty in diagnosing needle spots on weak or runty

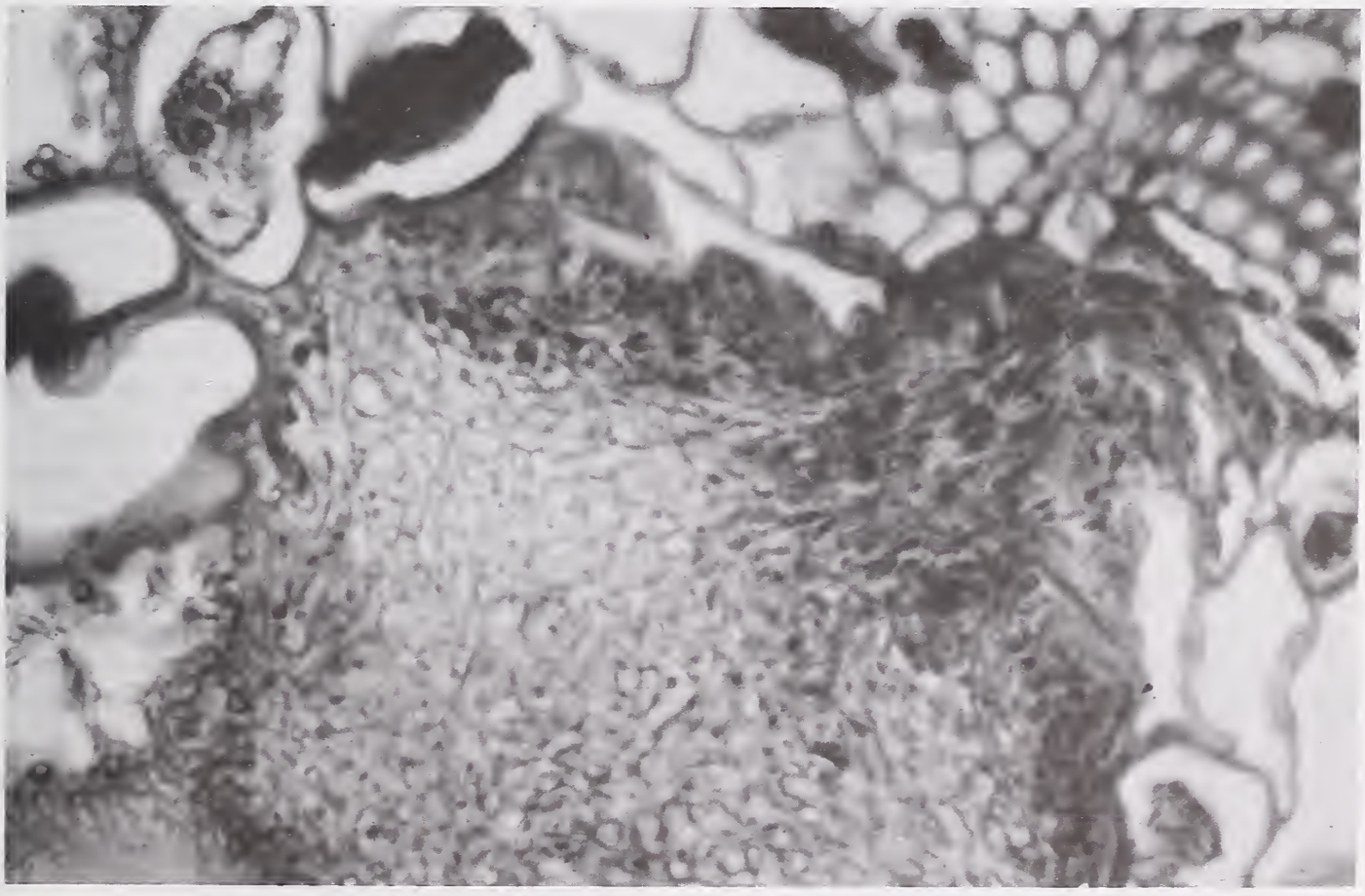


Figure 8.—Frozen section showing the massive pseudostroma of entwined *C. ribicola* mycelium usually found underlying a blister rust needle spot on *P. monticola*, about 11 to 12 months after inoculation.

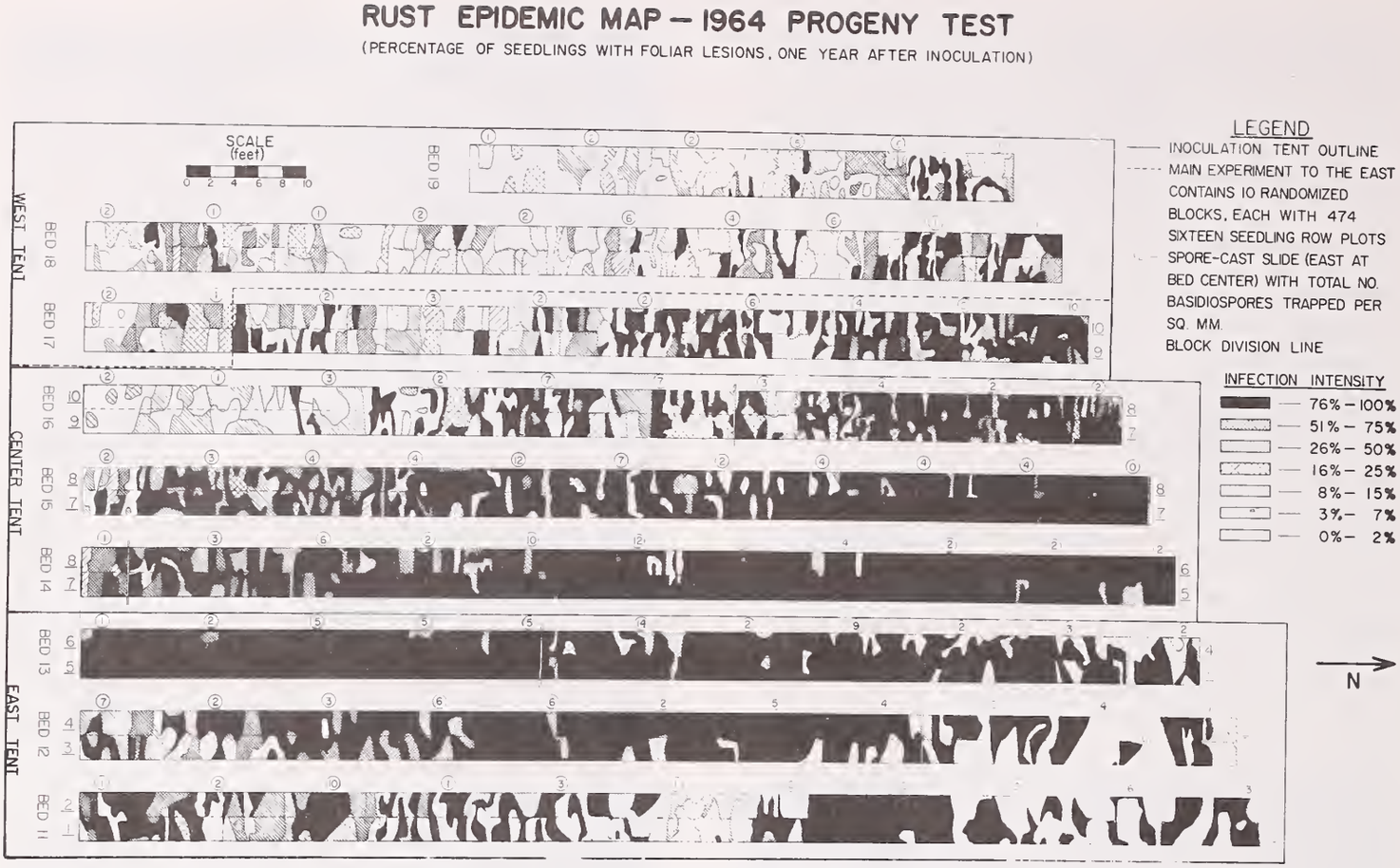


Figure 9.—Variation in percentage of seedlings infected with foliar lesions one year after artificial inoculation, 1964 progeny test.

seedlings, especially those with yellowish foliage. Often the runty seedlings had produced only one or a few bundles of second-year needles, and trying to distinguish lesions on the short and often discolored needles gave questionable results. This was the case with our foliage-lesion examinations on the normally more yellow-foliaged *P. strobus* progenies or on the somewhat yellowish-foliaged *P. monticola* × *P. strobus* hybrid progenies.

Needle spot inspection methodology, improved in several ways since the first (summer 1954) inspection, proceeded along two lines. First, each test seedling was rated either as spotted or spot-free. Second, each test progeny was scored on spot frequency—that is, the number of needle spots on a foliage sample involving a given number or lineal length of needles. Initially, we estimated spot frequency from a sample of 450 needles (contained in the 90 topmost 5-needle bundles) per progeny. But later, to eliminate differences in the total length of needles, we sampled the portions of the needles of the one to four topmost 5-needle bundles that lay inside a 3-inch (7.6-cm) diameter wire ring; this ring was centered about the seedling stem and held in place by a single, stiff wire leg poked into the ground.

During the shakedown period when we were first attempting to assess intensity of foliar infection, we tried unsuccessfully to adapt some cereal rust resistance technology to our tests. This was the use of subjective classes: spot-free = Class I; about 1 to 5 spots = Class II; about 6 to 25 spots = Class III; and so forth. This is similar to schemes still in use for rating severity of uredial infection on cereal leaves and stems. However, we found no statistical correlation between average spot-intensity class and the needle spot frequencies per foliage sample as outlined above. It should be pointed out here that our pine seedlings probably were far more variable in respect to size of foliar target than were the cereal seedlings. A given *P. monticola* seedling, in fact, might have more than 10 times the length or surface area of needles as might another; and one *P. monticola* progeny might have two to five times or more the length or surface area of needles as might another. Thus, the subjective classification schemes that held much promise for simplifying and shortening examinations had to be abandoned.

FOURTH YEAR, FOLIAR AND BARK INSPECTIONS

During the fourth year, we conducted a second inspection for foliar infection and a first inspection for bark infection. This was in July to early August, 21 to 22 months after artificial inoculation and 10 to 11 months after first natural inoculation. Seedlings were 4 years old.

Each test seedling was reinspected as follows:

1. For "old" needle-spots (presumably persistent from the previous year) on the artificially inoculated, second-year foliage still being retained on the seedlings.
2. For new needle-spots on the second-year needlage of previously uninfected seedlings (either latent from the artificial inoculation, or new from natural inoculation on the plot site).
3. For new needle spots on the seedling's third-year needlage that had to have come from natural inoculation.
4. For blister rust bark lesions (cankers) and any resistance reactions thereon.

Over the years it became apparent that natural inoculation, even from concentrations of planted *Ribes* spp. bushes, was at best a slow and irregular process, varying widely by year and outplanting site. For instance, with the 1952 progeny test, the 1955 examinations disclosed that there were 217 of 2,513 seedlings on the Elk Creek plot site that had new spots on 1954 foliage. These had to arise from natural inoculation because the 1954 foliage had not yet been produced when the seedlings were artificially inoculated in 1953. Meanwhile, on the Fernwood and Randolph Creek plot sites, numbers of naturally inoculated seedlings were 97 of 2,572 and 15 of 2,438, respectively. The continuing natural inoculation, however, did prove to be useful for "filling out" infection in those portions of the nursery beds where the level of seedling infection had been unsatisfactorily low. Nevertheless, it was abundantly evident that a progeny testing regime relying mostly on artificial inoculation was by far the more efficient process.

FIFTH AND SIXTH YEARS, BARK INSPECTION

We conducted the second and third inspections for bark infection in July and early August of the fifth and sixth years. This was 3 to 4 years after artificial inoculations, and after 2 to 3 years of natural inoculation. Seedlings were 5 to 6 years old. We looked for any blister rust bark lesions (cankers) and any resistance reactions thereon.

SEVENTH AND EIGHTH YEARS, BARK INSPECTIONS

Our fourth and fifth inspections for bark infection were in July and early August of the seventh and eighth years. This was 5 to 6 years after artificial inoculation, and after 4 to 5 years of natural inoculation. Seedlings were 7 to 8 years old.

Bark canker and resistance reaction examinations continued as in the preceding years. These inspections were only carried out for the four preliminary progeny tests because thereafter it became apparent that results changed very little after the third year of such inspection. Also, a final and confirmatory inspection was made in 1966, 10 to 13 years after artificial inoculation of seedlings 12 to 15 years old. This examination did not change the above conclusion.

RESULTS FROM THE PRELIMINARY PROGENY TESTS

Of the four preliminary tests sown between 1952 and 1955, the first (1952) contained the widest array and greatest number of controlled and wind-pollinated test progenies, received one of the more effective artificial inoculations, and provided the most meaningful long-term results. Thus, the results outlined here come almost entirely from this 1952 test. They are quite representative, however, of the other three tests.

For the 1952 test, results were obtained in the course of annual rust examination made between 1954 and 1958 (at test seedling age 3 to 8 years), followed by a confirmatory examination in 1966 (seedling age 15 years).

Through 1957, attrition due to nongerminable seeds, damped-off seedlings, and older seedlings dying from known or unknown causes other than blister rust, had reduced the number of seedlings in the average test progeny to 81. Thereafter, however, there were only minor seedling losses from such extraneous causes.

Genetic Variation in Foliar Infection

Because of experiences of Clinton and McCormick (1919), Spaulding (1922), York and Snell (1922), and other early workers with young *P. strobus* seedlings, we expected to see some blister rust foliage lesions about 1 year after artificial inoculation. We were unprepared, however, for the spectacular effects of such inoculation on 2-year-old *P. monticola* seedlings. Numerous large, vigorous, and many-needled seedlings bore literally hundreds of blister rust needle spots, often coalescent along their needles (fig. 7).

Across the entire 1952-sown test 10 to 11 months after artificial inoculation, we found an average of 177.5 needle spots per 450 needles sampled. This amounted to about one spot for each 2.5 needles, so that if the average seedling had 15 to 25 bundles of needles it probably also had about 30 to 50 needle spots.

Thus, the artificial inoculation had been a resounding success; but we wondered if we might not have seriously overexposed test seedlings to blister rust, perhaps eclipsing some of the forms of resistance that might be present.

The first evidences of blister rust resistance concerned parent-associated variation in (1) the percent of test progeny seedlings that exhibited needle spots, and (2) the number of needle spots found on a 450-needle sample of the seedlings in various test progenies. These two evidences of resistance are apparent in the summarized needle spotting data for the 1952 test (tables 3 and 4).

We could hardly wait to assemble such impressive tables as 3 and 4, but once we had, about the first thing we noted was how inefficient for characterizing the nature and extent of transmitted resistance had been our hit-or-miss mating scheme. Thus, selections such as parent tree numbers 19, 20, or 58 that had borne large numbers of control-pollinated strobili, plus a few other selections such as parents 25 or 30 that had borne phenologically early and abundant pollens, were overrepresented in the progeny test with 9 to 19 control-pollinated test progenies apiece. Meanwhile, other selections such as 10, 15, 23, 27, 28, 29, 34, 38, and 45 (almost 40 percent of the 23 selections tested) were underrepresented, having only 1 to 3 control-pollinated test progenies because they had borne few female strobili or phenologically late or scanty pollens.

Furthermore, mean parental line performances (here represented by the mean control-pollinated progenies of the next to the bottom lines of tables 3 and 4) were not comparable, for whenever there were more than 1 progeny in the parental line, the lineage, other than line parents, varied. For example, parents number 10 and 15 each were represented in the progeny test by 3 control-pollinated crosses. Yet 4 of the 6 crosses (19×10, 20×10, 15×1, and 15×30) were unrelated other than by line parents 10 and 15, and only 2 of the 6 crosses (25×10 and 15×25) were useful for estimating relative performance of the 2 parents. Thus, the mean control-pollinated progenies of parental lines 10 and 15 (respectively with 80.3 and 71.6 percent of their test seedlings bearing blister rust needle spots, next to bottom line table 3; or with 186.7 and 131.0 spots, next to bottom line table 4) could not be expected to appraise very well the relative ability of parents 10 and 15 to transmit resistance, either in respect to each other or to the other 21 parents in the test.

Table 3.—Percent of test progeny seedlings having one or more blister rust needle lesions on 1953, 5-needle-bundle foliage 11 months after artificial inoculation, 1952 progeny test[1]

Male parent	Female parent																							
	1	10	15	16	17	18	19	20	21	22	23	24	25	27	28	29	30	34	37	38	39	45	58	Wind
1			70	88			76	80		74						(74)	78		70[5]					
10							84	78[5]					79											
15	(70)												(72)				(73)							70
16	(88)						74	79	(62)		71		(90)				(72)						84	67
17							81		44	72	55		(74)				(71)		66				72	53
18							78						74				(73)						72	74
19	(76)	(84)		(74)	(81)	(78)	73	(79)	(39)	(73)		(78)[5]			(76)	(84)	(90)	(82)	(35)[5]	69	(89)	52	(70)	76
20	(80)	(78)[5]		(79)			79			(89)		(54)		81			(73)				(84)			69
21				62	(44)		39						60				66		(68)				56	32
22	(74)				(72)		73	89		54[5]			(84)			(88)	(77)						82	84
23				(71)	(55)																			64
24							78[5]	54													69		60	68
25		(79)	72	90	74	(74)			(60)	84							80[2]		75[5]				73	64
27								(81)																74
28							76																	66
29	74						84			88													71	
30	(78)		73	72	71	73	90	73	(66)	77			80[2]				56[5]		(80)		83		74	77
34							82																	
37	(70)[5]				(66)		35[5]		68				(75)[5]				80						67	
38							(69)																	
39							89	84				(69)					(83)				68		64	44[5]
45							(52)																	
58				(84)	(72)	(72)	70		(56)	(82)		(60)	(73)			(71)	(74)		(67)		(64)		81	72
Wind[3]			(70)	(67)	(53)	(74)	(76)	(69)	(32)	(84)	(64)	(68)	(64)	(74)	(66)		(77)				(44)[5]		(72)	57[4]
No. control crosses	8	3	3	8	8	4	19	9	7	9	2	4	10	1	1	4	14	1	7	1	6	1	13	16
Average percent infection	76.2	80.3	71.6	77.5	66.9	74.2	72.9	77.4	56.4	77.0	63.0	65.2	76.1	81.0	76.0	79.2	74.7	82.0	65.8	69.0	76.2	52.0	71.2	65.9

Grand average for control pollinated crosses: 72.2

[1]The mean test progeny contained an average of 81 test seedlings distributed over all 9 randomized blocks. The number of test progenies (160) shown below in the body of the table, represents an artificial doubling of the 80 progenies actually tested; the values for the reciprocal crosses are added in parentheses.
[2]In this case the reciprocal crosses actually were made, the value here being the average of the 2 crosses.
[3]These values for wind-pollinated progenies are not included in the average percentages.
[4]The cross wind × wind is the average value for the 5 control lots.
[5]Based on a total of only 14 to 26 seedlings, these represented in only 2 to 6 of the 9 randomized blocks.

Table 4.—Number of blister rust needle lesions on a 450-needle sample of 1953 foliage 11 months after artificial inoculation, 1952 progeny test[1]

Male parent	Female parent																							
	1	10	15	16	17	18	19	20	21	22	23	24	25	27	28	29	30	34	37	38	39	45	58	Wind
1			120	202			116	163		258						(153)	89		49[5]					
10							256	102[5]					202											
15	(120)												(155)				(118)							128
16	(202)						178	277	(232)		162		(239)				(217)						232	187
17							178		57	186	89		(132)				(216)		80				53	74
18							173						148				(209)						77	253
19	(116)	(256)		(178)	(178)	(173)	128	(267)	(37)	(163)		(101)[5]			(131)	(208)	(402)	(102)	(113)[5]	249	(190)	135	(77)	252
20	(163)	(102)[5]		(277)			267			(350)		(176)		235			(260)				(251)			183
21				232	(57)		37						101				137		(167)				77	74
22	(258)				(186)		163	350		333[5]			(233)			(269)	(262)						154	260
23				(162)	(89)																			194
24							101[5]	176													284		148	95
25		(202)	155	239	132	(148)			(101)	233							162[2]		131[5]				77	211
27								(235)																406
28							131																	122
29	153						208			269													90	
30	(89)		118	217	216	209	402	260	(137)	262			(162)[2]				510[5]		(205)		359		245	87
34							102																	
37	(49)[5]				(80)		113[5]		167				(131)[5]				(205)						133	
38							(249)																	
39							190	251				(284)					(359)				247		269	109[5]
45							(135)																	
58				(232	(53)	(77)	77		(77)	(154)		(148)	(77)			(90)	(245)		(133)		(269)		152	147
Wind[3]			(128)	(187)	(74)	(253)	(252)	(183)	(74)	(260)	(194)	(95)	(211)	(406)	(122)		(87)				(109)[5]		(147)	142[4]
No. control crosses	8	3	3	8	8	4	19	9	7	9	2	4	10	1	1	4	14	1	7	1	6	1	13	16
Average number spots/ 450 needles	143.8	186.7	131.0	217.4	123.9	151.8	168.6	231.2	115.4	245.3	130.5	177.2	158.0	235.0	131.0	180.0	242.2	102.0	125.4	249.0	266.7	135.0	137.2	173.9

Grand average: 181.1

[1]The mean test progeny contained an average of 81 test seedlings distributed over all 9 randomized blocks. The number of test progenies (160) shown below in the body of the table, represents an artificial doubling of the 80 progenies actually tested; the values for the reciprocal crosses are added in parentheses.
[2]In this case the reciprocal crosses actually were made, the value here being the average of the 2 crosses.
[3]These values for wind-pollinated progenies are not included in the average percentages.
[4]The cross wind × wind is the average value for the 5 control lots.
[5]Based on a total of only 14 to 26 seedlings, these represented in only 2 to 6 of the 9 randomized blocks.

Nevertheless, it was encouraging to note in table 3 that seedling progenies of a few parents appeared rather consistently to be resisting foliar infection. The 4 to 8 progenies each of parents 17, 21, 24, and 37 seemed to stand out in this respect. Conversely, other parents such as numbers 1, 16, 20, 22, 25, and 39 rather consistently produced progenies that appeared to be quite susceptible. Thus, there appeared to be general combining ability (GCA) for both parts of the host's resistance: susceptibility system. There also appeared to be additivity in the system, for when we had crossed parents both notable for producing progenies low (or high) in percent of needle-spotting, then the resulting progenies averaged even lower (or higher) in respect to the grand mean test progeny than had the parental line mean progenies.

Finally, some test progenies (for instance 19 × 21 or 19 × 37) seemed to demonstrate specific combining ability (SCA) for resistance to needle-spotting, since the values for percent spotted (39 and 35 percent, respectively) were well below those expected on the basis of the average progenies of parents 19, 21, or 37.

These promising features of the resistance system were emphasized even more by data on needle spot frequency (table 4). The data show that 3 of the 4 parents that exhibited good GCA for resistance in respect to needle spotting (specifically numbers 17, 21, and 37), plus parents 1 and 58, exhibited GCA for low frequencies per 450-needle samples. Meanwhile, 4 of the 6 parents that exhibited GCA for susceptibility (specifically numbers 16, 20, 22, and 39) also exhibited GCA for high frequencies per 450-needle sample. Again, progenies such as 19 × 21 (37 spots per 450 needles) and 58 × 25 (77 spots per 450 needles) seemed to demonstrate SCA for low frequency. Again there appeared to be additivity in the system (compare the 8 chance crosses including low spot frequency parents numbers 1, 17, 21, 27, and 58; that is, crosses 37 × 1, 37 × 17, 21 × 17, 21 × 37, 58 × 17, 58 × 21, 58 × 37, and 58 × 58). Each of the crosses exhibited lower than average spotting frequency within parental lines, and especially against the grand mean control-pollinated progeny.

In his review of this paper Tony Squillace pointed out the possibility that spotting frequency might simply be inherited. He noted that when, from the data of table 4, he assigned the genotypes homozygous dominant susceptible (SS, to certain parents such as numbers 10, 16, 20, and 27), homozygous recessive resistant (ss, to certain parents such as 17, 21, and 58), or heterozygosity (Ss, to certain parents such as 1, 15, 19, and 37), then the various crosses of table 4 showed an orderly decrease in spotting as one progressed from the cross SS × SS to the cross ss × ss, as below. He computed average degree of dominance as near 0.75.

Proposed genotype of cross	Number of crosses	Average number of spots/450 needles
SS × SS	9	257.0
SS × Ss	22	213.2
SS × ss	9	208.9
Ss × Ss	14	131.1
Ss × ss	13	106.6
ss × ss	3	62.3

This interested us because Hoff and McDonald (1980) had tested similar analyses and reported similar findings for a later progeny test. They stated that while a single (major) gene hypothesis did not fit the resistance-susceptibility system, nevertheless, single incompletely dominant gene hypothesis fit the system best.

Tables 3 and 4 also brought out the anomalous performance of many wind-pollinated progenies of rust-free parental selections. Of seedlings needle-spotted in the 16 wind-pollinated progenies of table 3, the average percent was 65.9, while seedlings of the 59 control-cross-pollinated progenies of the same 16 parents averaged 72.4 percent needle-spotted. And the average number of needle spots per 450 needle sample for the 16 wind-pollinated progenies of table 4 was 173.9, while the 59 control-pollinated progenies of the same 16 parents averaged 178.6 per 450 needles.

Thus, it appeared that on the average the wind-pollinated progenies of rust-free selection were slightly more resistant than progenies from controlled crosses between two rust-free selections. This was counter to our expectations. We assumed that with mainly outcrossing and panmixis under wind-pollination, the rust-free selections would have been pollinated by a variety of susceptible neighbors. An unexpectedly great number of selfs in the wind-pollinated progenies was the only logical theory we could advance to explain these aberrancies. However, the height difference (only 0.009 ft or 2.7 mm) between the average wind- and control-pollinated progenies of the 16 parents belied any such theory.

The apparently good performance of the five ordinary nursery control progenies was even more surprising. We thought these materials were the most susceptible in the progeny test, yet they proved (in tables 3 and 4) to contain over 15 percent fewer spotted seedlings and to bear almost 40 fewer spots per 450 needles than the grand mean control-pollinated test progeny. However, there was an acceptable explanation for this apparently low susceptibility. Four of the five original control lots had germinated at levels of only 1 to 5 percent in the nursery beds in 1952, and the fifth lot, less than 15 percent. The four worst lots were replaced completely by transplanting later-germinating and thus somewhat younger and smaller seedlings from Savenac Nursery, and the 55 percent of vacant plant bands of the fifth lot were replaced by transplanting from a standby bed of the same control lot.

Two years later when test seedlings were inoculated (in the fall before needle spotting was assessed in the 3-year -old seedlings) these 5 control lots averaged only about two-thirds the height, about 2.01 inches (5.1 cm), of the average control-pollinated progeny, or about 2.99 inches (7.6 cm). In general they also appeared to be weaker and more runty. Paired "t" test showed that the shorter and less vigorous transplants were indeed a different population than normal seedlings; they were significantly (1 percent level) less infected and less heavily spotted than normal seedlings, following the old rule what weak plants are relatively poor suscepts for obligate parasites.

Thus, we thought that the aberrant performance of wind-pollinated and control progenies would disappear with time as latent infections from the 1953 artificial inoculation and new infection from 1954 or later natural inoculations began to appear.

Information in tables 3 and 4 made it apparent that for the same money and work we would have been better off and further along had we consistently employed some factorial mating scheme (for instance, a scheme wherein each rust-free parental selection had been crossed with 4 to 8 other "tester" selections). Alternatively, was it possible to simulate such a factorial mating scheme, using certain of the matings we had entered as test progenies in the 1952 progeny test? Luckily, such a simulation appeared to be possible using the five most frequently crossed selections (numbers 19, 20, 25, 30, and 58) as

"testers" and the nine other frequently crossed selections (numbers 1, 16, 17, 18, 21, 22, 24, 30, and 37) as the other parents. In this simulated 5 × 9 factorial cross there would be only 9 (of 45) cells, or crosses, missing, and each of the 9 parents would be represented by at least three of the five tester matings (see table 5). Therefore, we decided to show and analyze all of the test results according to this incomplete, 5 × 9 factorial cross (as in tables 5–10).

We also decided to present results on foliar and bark resistance only after the blister rust disease had mostly run its course on the test progenies. With the 1952 progeny test, fairly conclusive results could have been obtained 4 years after the effective 1953 artificial inoculation. However, another, even more conclusive examination had been made in 1966; therefore, the results on tables 5–12 are given as of that year.

Variation in Percent of Rust-Free Seedlings

In time, rust from the artificial inoculation and the continuing natural inoculations almost blanketed the progeny test seedlings (tables 5 and 6). Even so, most the the more lightly infected parental lines noted in table 3 (17, 21, 24, and 37) remained the least infected through 1966, although by then percentages of rust-free seedlings had become very small. And the more heavily infected parental lines of table 3 (16, 20, 22, 30, and 39) approached 100 percent infection by 1966.

Thus, while in some families of table 5 there seemed to be a basis for complete freedom from rust, we were unable to substantiate such a belief. In fact, the proportions of seedlings exhibiting this character were so small that we hesitated to regard the rust-free seedlings as other than chance "escapes" from infection.

The passage of time also allowed us to further explore the perplexingly low susceptibility of wind-pollinated and ordinary nursery control seedlings that had been recorded one year after artifical inoculation (tables 3 and 4). Our expectation was that these apparently low susceptibilities would disappear with time, as indeed they did (table 6). At the bottom, right corner of table 6 are two sets of four percentages of infection that speak to this point. Note that through 1966 percentages of infection in the average wind-pollinated and control progenies caught up with or surpassed those in the average control-pollinated progeny, the control progenies by then being the most susceptible (98.6 percent infected). Progress of the rust epidemic as it blanketed the control-pollinated progenies is shown for individual progenies of the 5 × 9 factorial mating in the body of table 6.

Table 5.—Percent of infection with increasing progeny age (or by date of inspection) in an incomplete 5 × 9 factorial mating from the 1952 progeny test, through 1966[1]

Other parent	Tester parent 19				20				25				30				58				Family averages			
	1954	1955	1957	1966	1954	1955	1957	1966	1954	1955	1957	1966	1954	1955	1957	1966	1954	1955	1957	1966	1954	1955	1957	1966
1	76	84	94	95	80	84	94	98					78	90	93	94					78.0	86.0	93.7	95.7
16	74	90	94	98	79	85	98	99	90	94	96	98	72	91	97	97	84	88	98	99	79.8	89.6	96.6	98.2
17	81	91	96	96					74	86	92	94	71	87	94	94	72	86	91	91	74.5	87.5	93.2	93.8
18	78	90	97	97					74	91	97	98	73	92	99	99	72	84	94	94	74.2	89.2	96.8	97.0
21	39	73	93	93					60	82	97	99	66	84	96	100	56	79	93	94	55.2	79.5	94.8	96.5
22	73	84	94	96	89	93	99	99	84	96	98	100	77	90	96	97	82	91	96	97	81.0	90.8	96.6	97.8
24	78	95	100	100[2]	54	80	90	92									60	78	86	90	64.0	84.3	92.0	94.0
37	35	54	81	81[2]					75	75	79	79[2]	80	91	94	99	67	82	91	95	64.2	75.5	86.2	88.5
39	89	92	94	95	84	93	99	99					83	87	96	98	64	78	86	91	80.0	87.5	93.8	95.8
Family averages	69.2	83.7	93.7	94.6	77.2	87.0	96.0	97.4	76.2	87.3	93.2	94.7	75.0	89.0	95.6	97.2	69.6	83.3	91.9	93.9	73.4	86.1	94.1	95.6
													Averages of wind-pollinated progenies[3]								64.7	80.9	88.4	95.2
													Averages of 5 control progenies								59.6	79.9	93.1	98.6

[1]Unless noted under footnote 2, below, the test progenies averaged 81 seedlings distributed across all 9 randomized blocks.
[2]In all four years, based on a total of from 14 to 41 seedlings represented in 2 to 6 of the 9 randomized blocks.
[3]These averages are based on 12 of the 14 parents in the 5 × 9 factorial.

Table 6.—Percentage of completely rust-free seedlings in the test progenies of an incomplete 5 × 9 factorial mating from the 1952 progeny test, through 1966[1]

Other parent	Tester parent 19	20	25	30	58	Family averages
1	5.1	3.5		3.4		4.0
16	2.3	1.2	2.3	3.4	1.1	2.1
17	5.9		6.9	5.7	7.8	6.6
18	3.4		2.3	1.1	5.0	3.1
21	6.8		1.1	1.1	5.6	3.6
22	4.4	.0	.0	3.3	3.4	2.2
24	2.4[2]	7.7			7.9	6.0
37	19.2[2]		21.4[2]	1.3	5.7	11.9
39	4.6	1.3		2.1	9.1	4.3
Family averages	6.0	2.7	5.7	2.7	5.8	Grand average 4.6

[1]Unless noted under footnote 2, below, the test progenies averaged 81 seedlings distributed across all 9 randomized blocks.
[2]Based on a total of from 14 to 41 seedlings represented in 2 to 6 of the 9 randomized blocks.

Genetic Variation in Needle Lesion Frequency

The data on needle-spot frequencies arrayed for the same 5 × 9 factorial cross (table 7) proved to be more encouraging. Again, they followed the trends already outlined for table 4, but the differences between parental lines (families) were great enough and performance of progenies within parental lines consistent enough so that the genetic implications were clear. Thus, families of parents such as selection numbers 17, 19, 21, and 58, and possibly 24 and 37, contained progenies consistently below the grand average progeny in numbers of needle spots per 450 needles. Conversely, families of parents such as selection numbers 16, 20, 22, 30, and 39 contained progenies consistently above the grand average in numbers of needle spots. Presence of general combining ability (GCA) both for low and high needle spot frequency was obvious. Furthermore, prog-

Table 7.—Number of blister rust needle spots found on a sample of 450 secondary needles in the test progenies of an incomplete 5 × 9 factorial mating from the 1952 progeny test[1]

Other parent	Tester parent					Family averages
	19	20	25	30	58	
1	116	163		173		150.7
16	178	277	239	217	232	228.6
17	178		132	216	53	144.8
18	173		148	209	77	151.9
21	37		101	137	77	88.0
22	163	350	233	262	154	232.4
24	101[2]	176			148	141.7
37	113[2]		131[2]	205	133	145.5
39	190	251		359	269	267.3
Family averages	138.8	243.4	164.0	222.2	142.9	Grand average 177.0

[1]Unless noted under footnote 2, below, the test progenies averaged 81 seedlings distributed across all 9 randomized blocks.
[2]Based on a total of from 14 to 41 seedlings represented in 2 to 6 of the 9 randomized blocks.

enies from matings between parents both of which produced characteristically lightly spotted lines, or heavily spotted lines, demonstrated the decided additivity present in the resistance: susceptibility systems. The inference could be drawn that identical resistance (or susceptibility) genes were present in several of the parental selections.

These were heady conclusions for us, and they delighted our cooperators and steering committee[8] members as well. We would have been even more delighted had we realized at the time that low needle lesion frequency probably was a uniform or horizontal resistance factor that might, characteristically, be more stable.

Genetic Variation Expressed by the "Spots-Only Syndrome"

Within a year our spirits received another boost as we began accumulating evidence of another form of foliar resistance. By 1955, it was becoming apparent that certain test seedlings that had borne blister rust needle spots thereafter failed to develop either suspect bark reactions or definite blister rust bark cankers. This phenomenon, outlined in table 8, soon became known as the "spots-only syndrome." In certain families of progenies (notably those of parents 17, 19, 20, 22, 24, and 58) average percentages of seedlings surviving rust attack due to the spots-only syndrome ranged up to 11 percent. Individual progenies in these lines ranged up to almost 17 percent survival due to the spots-only syndrome, while the grand average progeny had about 6 percent of its seedlings surviving. Again, GCA and additivity were strong features of the genetic system.

Table 8.—Percentages of progeny seedlings in the incomplete 5 × 9 factorial cross that had blister rust needle lesions but thereafter developed no bark reactions or cankers and thus survived, 1952 progeny test, through 1966[1,3]

Other parent	Tester parent					Family averages
	19	20	25	30	58	
1	9.3	2.4		2.3		4.7
16	4.6	4.7	0.0	1.8	3.5	2.9
17	11.2		6.2	9.8	16.9	11.0
18	1.2		1.2	7.9	1.2	2.9
21	9.1		1.1	1.1	2.3	3.4
22	11.6	11.4	6.7	2.3	11.6	8.7
24	5.0[2]	13.9			13.4	10.8
37	4.0[2]		9.1[2]	.0	4.8	4.5
39	8.4	1.4		2.2	1.2	3.3
Family averages	7.2	6.8	4.0	3.4 [1]	6.9	Grand average 5.7

[1]Unless noted under footnote 2, below, the test progenies averaged 81 seedlings distributed across all 9 randomized blocks.
[2]Based on a total of from 14 to 41 seedlings represented in 2 to 6 of the 9 randomized blocks.
[3]Percentages were computed after having reduced the numbers of seedlings tested in the various test progenies by the numbers of rust free seedlings (the seedlings of table 6).

[8]See appendix for anecdote.

Genetic Variation in Bark Resistance

We had been alerted to expect resistance reactions seated in the host bark by Riker and others (1949, 1953) and by Struckmeyer and Riker (1951), workers who reported the "corking-out" of established blister rust bark cankers in *P. strobus*. Sure enough, by 1955 to 1957 we began seeing various bark resistance reactions in our *P. monticola* test seedlings. With *P. monticola*, however, the host seedling's elimination of established infection took several forms. These included:

1. Rapid death and collapse of the infected bark tissues in the area of a young canker centered on the base of a needle bundle, usually under 0.25 inch (0.65 cm) diameter (fig. 10).

Figure 10.—These areas of previously infected bark centered about needle bundle bases collapsed and died so rapidly that we often missed the typical, orange bark discoloration associated with blister rust cankers.

2. Death and collapse of infected bark tissue but only as a ring around a canker situated as in (1) above.
3. More extensive bark reactions, most often centered about a needle bundle base with the disturbed bark tissue originally supporting rust mycelium, but the reaction area never assuming the orange-discolored, spindle-shaped canker symptoms typical of normal bark infections, nor later supporting the normal pycnial and aceial signs of the rust, usually 0.50 to 1.0 inch (1.3 to 2.5 cm) in length (fig. 11).
4. Still larger and rougher surfaced bark reactions that once had shown typical symptoms of the disease or signs of the rust fungus, but where the infected bark of the canker had been walled in by marginal wound phellogens—that is, "corked-out" in the Struckmeyer and Riker (1951) sense (fig 12).

Figure 11.—This bark reaction never showed the typical, outward symptoms or signs of the blister rust disease. It remained under 1 inch (2.5 cm) in length and eventually disappeared.

Figure 12.—This bark reaction once showed the typical orange discoloration of an active blister rust bark canker. Later the infected area of bark was sealed off and died inside rings of wound phellogens.

However, we could discern no patterns of parental performance in respect to seedling survival associated with these four kinds of bark resistance reaction, so we lumped all survival associated with the reactions in table 9. These were the most encouraging results we had seen emerge. Six of the 36 test progenies of the incomplete 9 × 5 factorial cross contained 20 percent to 30 percent of seedlings that survived rust attack, apparently due to their bark resistance. The parental lines 17, 19, 22, 24, and 58 were outstanding in bark resistance, the family average progenies containing from almost 4 percent to 12 percent more seedling survivors than did the grand mean progeny. Again, GCA, SCA, and additivity appeared to be features of the resistance system.

Table 9.—Percent of progeny seedlings in the incomplete 5 × 9 factorial cross surviving due to bark resistance reactions 1952 progeny test, through 1966[1,3]

Other parent	Tester parent					Family averages
	19	20	25	30	58	
1	12.1	1.2		2.4		5.2
16	2.4	2.5	0.0	.0	6.0	2.2
17	30.4		15.0	14.4	24.7	21.2
18	12.1		1.2	3.5	3.6	5.1
21	20.2		1.1	3.5	8.6	8.4
22	22.6	30.5	3.6	5.9	22.9	17.1
24	13.4[2]	8.2			27.2	16.3
37	10.3[2]		.0[2]	3.9	9.0	5.8
39	13.9	.0		2.2	6.3	5.6
Family averages	15.3	8.5	3.5	4.5	13.5	Grand average 9.6

[1]Unless noted under footnote 2, below, the test progenies averaged 81 seedlings distributed across all 9 randomized blocks.
[2]Based on a total of from 14 to 41 seedlings represented in 2 to 6 of the 9 randomized blocks.
[3]Percentages of surviving seedlings were computed removing seedlings that had survived because they never became infected (as in table 6), or because of the spots-only syndrome (as in table 8).

Genetic Variation in Seedling Survival

Finally by 1957, 4 years after artificial inoculation at seedling age 6 years, we were ready to estimate the total percentages of progeny seedlings that survived rust exposure or attack. This figure, after all, was the most important in respect to the practical utility of the tested, first-generation stocks. As with tables 5 to 9, 1966 results are given here, but they had changed little since 1957. These total percentages of survival due to all resistance factors are given in table 10. Individual progenies contained from less than 2.5 percent to more than 41 percent surviving seedlings, with the grand average progeny containing almost 18 percent of survivors, or 14 percent more survivors than the controls—encouraging numbers. Five of the 14 parents (5 testers and 9 other parents) tested in the incomplete factorial cross (numbers 17, 19, 22, 24, and 58) seemed to be exhibiting good GCA for resistance. Their average progenies ranged from about 5 percent to 15 percent higher in seedling survival than the factorial's grand mean progeny (with 17.9 percent survival).

Table 10.—Percent of progeny seedlings surviving due to all kinds of resistance reactions of the host foliage and bark in an incomplete 5 × 9 factorial cross, 1952 progeny test, through 1966[1,3]

Other parent	Tester parent 19	20	25	30	58	Family averages
1	24.1	7.1		7.8		13.0
16	9.1	8.1	2.3	5.1	10.2	7.0
17	40.0		25.2	26.3	41.2	33.2
18	15.7		4.5	12.2	10.0	10.6
21	32.2		3.3	5.6	15.5	14.2
22	26.7	36.4	10.0	18.6	33.6	25.1
24	19.5[2]	26.9			40.5	29.0
37	30.6[2]		28.5[2]	5.2	18.1	20.6
39	20.6	2.6		6.3	15.9	11.4
Family averages	24.3	16.2	12.3	10.9	23.1	Grand average 17.9
				Average of 5 controls		4.1

[1]Unless noted under footnote 2, below, the test progenies averaged 81 seedlings distributed across all 9 randomized blocks.
[2]Based on a total of from 14 to 41 seedlings represented in 2 to 6 of the 9 randomized blocks.
[3]Percentages were computed using total number of surviving seedlings over the total number of seedlings tested—that is, there were no progressive reductions in the numbers of test seedlings as in tables 8 and 9.

Table 11.—Percentage of seedlings surviving in test progenies of parents exhibiting general combining ability for resistance, 1952 progeny test, through 1966[1]

Male parent	Female parent 17	19	22	24	58	
17		40.0	38.6		41.2	
19	(40.0)		(26.7)	(19.5)[2]	(24.4)	
22	(38.6)	26.7			33.6	
24		19.5[2]			40.5	
58	(41.2)	24.4	(33.6)	(40.5)		
Family averages	39.9	27.6	33.0	30.0	34.9	Grand average 33.1
				Average of 5 controls		4.1

[1]Test progenies contained on the average of 81 seedlings distributed across all 9 randomized blocks; the number of progenies is artificially doubled by entering values for the reciprocal crosses, in parentheses.
[2]Based on only 41 seedlings in 6 of the 9 randomized blocks.

Table 12.—Infection and survival of test seedlings in 6 self-pollinated progenies, 1952 progeny test, through 1966

Parent	Number seedlings tested	Percent of seedlings infected 1954	1955	1957	1966	No. spots per 450 needles, in 1954	Percent survival by resistance-reaction categories: Never infected	Needle spots only[1]	Bark reactions[1]	Total
19	90	73	90	96	96	128	4	7	30	38
20	8[2]	38	50	62	62	50	38	--	--	38
22	26[2]	54	79	83	90	333	10	18	29	47
30	18[2]	56	83	89	94	510	6	7	7	19
39	65	68	78	83	95	247	5	2	10	17
58	90	81	86	93	96	152	4	9	32	42

[1]Percentages were computed removing surviving seedlings from previous 1 or 2 columns from total numbers of seedlings tested.
[2]Represented by only 8 to 26 seedlings in only 2 to 4 of the 9 randomized blocks, otherwise by an average of 82 seedlings in all randomized blocks.

Level of Survival in "GCA-F_1" and S_1 Progenies

An outstanding and encouraging result was the survival in progenies of parents that both expressed GCA for resistance. Inadvertently we had produced eight such GCA-F_1 progenies from crosses among the five GCA trees, (parents such as numbers 17, 19, 22, 24, and 58). We found a 33.1 percent average level of surviving seedlings in the eight progenies. This was almost 30 percent higher than in the control progenies (table 11).

There were five GCA trees found in the 5 × 9 factorial cross, or over one-third of the 14 trees involved. However, a perusal of the rest of the 1952 progeny test data (including 62 more control-pollinated plus 16 wind-pollinated progenies) showed that the proportion of GCA trees was probably nearer to 1 out of 4 rust-free trees.

Also from the 1952 progeny test came information on the resistance of S_1 (self-pollinated) progenies (table 12). Here, performance of the larger and more reliable progenies followed that of the cross-pollinated progenies discussed above. There were small increments of resistance coming from several foliar and bark resistance factors, together accumulating to the point where about 20 to 40 percent of the self-pollinated seedlings survived intense exposure to the rust.

OTHER RESEARCH ON *PINUS MONTICOLA*

The BEPQ Office of Blister Rust Control and the author had been assigned the foregoing research, or developmental work, on blister rust resistance. Meanwhile, Tony Squillace of the Northern Rocky Mountain (now Intermountain) Forest and Range Experiment Station had been conducting a less well-staffed and financed program investigating *P. monticola* variation in respect to other qualities. In practice, Tony and I worked closely and published together on both phases of the work.

By 1954, we had produced evidence of significant correlation between height growth of *P. monticola* parents and F_1 progenies 85 ($r = 0.30$ to 0.80) (Squillace and Bingham 1954). Shortly thereafter we had detected and reported what appeared to be localized, site-associated, ecotypic variation, as well as elevation-associated variation in *P. monticola* height growth and seed germination (Squillace and Bingham 1958a). We had found and reported for self-pollinated *P. monticola* a 50 percent reduction in filled seed yield per cone, a 7 to 13 percent reduction in seed germinability, and an 11 to 21 percent reduction in early height growth (Bingham and Squillace 1955). We also described some of the phenological features of "flowering" in the species (Bingham and Squillace 1957). Also, Squillace had investigated within-tree variation in cone characters, seed yield, and seed weight of *P. monticola* (Squillace 1957), and he had installed a number of flower induction studies with the species. Lastly, we had assembled a great deal of raw data on cone and seed yields of young *P. monticola* trees.

Thus, by 1957 we had learned a little about the genetics of *P. monticola* aside from blister rust resistance, as well as a fair amount concerning the species' reproductive biology. Nevertheless, we needed to know a great deal more concerning (1) ecotypic and altitudinal variation as affecting plantings of improved *P. monticola* strains, and (2) seed orchard management for the species.

1957 — OUR YEAR OF DECISION

By late summer 1957, the positive and encouraging results on transmission and extent of blister rust resistance in the F_1 progenies of the 1952 progeny test had become so firm, and newer results from the 1953 to 1955 progeny tests so supportive, that we researchers could draw some fairly safe, if broad, conclusions concerning the nature, extent, and utility of blister rust resistance in Inland Empire *P. monticola*. Those conclusions were:

1. The apparent blister rust resistance that had been isolated by natural selection in rare, rust-free *P. monticola* trees in rust-decimated natural stands was indeed real, and it was under strong genetic control.

2. Crosses among the rust-free parent selections had produced F_1 progenies, and performance of these progenies showed that in all probability there were several, to many, resistance-genes in the overall resistance system. Effects of these genes were visible as a succession of host resistance reactions that occurred over 3 or more years as the rust spread first to foliage and later to bark.

3. Apparently many of the same resistance-genes occurred in the genotypes of different rust-free parental selections, for many of the same or similar resistance reactions occurred in F_1 progenies representing different parental selections.

4. There was little evidence that single, major (dominant or recessive) genes were present in the resistance system; rather, a seemingly classic picture of quantitative inheritance of resistance had emerged. Instead of the 25, 50, or 100 percent increments of resistance expected in progenies under single major-gene-controlled inheritance, we experienced much smaller increments associated with each resistance reaction. Polygenic inheritance, incomplete dominance, or some other form of inheritance of resistance was suggested.

5. Both general combining ability (GCA) and specific combining ability (SCA) were found in the resistance system, with GCA being a prominent feature. About one-fourth of the rust-free parents produced parental lines of F_1 progenies wherein most, or all, of several progenies were above average in resistance (that is, the parents exhibited GCA for resistance). And when these GCA parents were perchance mated, they produced noteworthy GCA-F_1 progenies in which an average of about 30 percent more of the F_1 seedlings survived intense, artificial and natural exposure to the rust than did control seedlings.

Thus, in 1957 we researchers and our administrators were faced with our first major policy and financial decision on whether, and how, to go ahead toward mass production of blister rust resistant planting stock. Based on the results and conclusions outlined here, the decision to go ahead was immediate and unanimous. But we reserved for further study detailed and technical questions such as: (1) level of resistance required for practical planting, (2) time available to secure that level of resistance, (3) stratigies we might be able to incorporate in the program as "insurance" against new or different pathogenic rust races, (4) new research facilities that would be needed, and (5) the funding and staff required to do the job.

The question of any new laboratory, greenhouse, nursery, and other facilities was not one we were allowed to ponder long. A chance, early fall 1957 visit by Forest Service Washington Office inspectors—happily coming at a time and place almost ideal for demonstrating extent of resistance—led within 2 weeks to construction funds for a research facility and tacit approval for increased R&D budgets and staff.[9] Planning and bid letting for the facility were completed by spring 1958, and construction was completed by that fall.

1958 to 1959 — A PLANNING AND TRANSITION PERIOD

We spent most of 1958 and 1959 winding up the four preliminary progeny tests, planning and establishing a new resistance research facility, and deciding the directions and priorities of new R&D work.

Decisions on directions and priorities were handled with the help of a Steering Committee for Blister Rust Resistance Research.[8] Together we made sometimes arbitrary assumptions and decisions just to get on with the work. Those assumptions and decisions were:

[8] See appendix for anecdote.
[9] See appendix for anecdote.

1. With the funds, staff, and time available, we wanted to secure plantable resistant stocks well before the year 2000. If this time limit could not be met, a reappraisal of the entire R&D program would be undertaken.

2. Some level of resistance substantially above the 30 percent survival observed for GCA-F_1 progenies under intense artificial inoculation would be necessary before resistant nursery stock could be considered plantable. Within the time limits of (1) above, this arbitrary decision meant: (a) use of GCA-F_1 stock, if 15 to 25-year tests showed the field level of resistance to be well above 30 percent survival; or (b) use of F_2 stocks bred from resistant F_1 seedlings, if level of survival under artificial inoculation was well above 30 percent.

3. To provide some genetic breadth against pathogenic variation in *C. ribicola*, as well as against inbreeding depression of growth in seed orchard stocks, we would have to substantially broaden the genetic base of rust-free parents entered in the program. Considering the money, staff, and time available, a 400-tree base would seem to be a realistic goal.

4. Primary selection would be for GCA for resistance. Thus, with only one in four parents embodying GCA, the 400-tree base would be reduced by selection to about 100 GCA trees. Then, to prevent maladaptation of planting stock, the 100-GCA-tree base probably would have to be further subdivided among elevational-zone orchards. This would reduce the base to an extent precluding improvement of any trait other than blister rust resistance.

5. There were about 2 million acres of potential white pine lands in the Inland Empire, roughly half of which fell in the better white pine site indexes. In these better lands, clearcutting and wildfire together could be expected to provide only about 10,000 to 20,000 acres per year for planting. Planting would be restricted to the better white pine lands with a spacing after rust losses not to exceed 15 ft by 15 ft (4.5 m by 4.5 m), or about 200 trees per acre (about 500 per ha).

Finally, before embarking on a R&D program at the new Genetics Center, we scientists added a few observations and recommendations of our own, mainly concerning improved technology for progeny testing. These recommendations were:

1. Use of a single, heavy, artificial inoculation at rust-sensitive seedling age 2 years had proved to be highly efficient for rapid and thorough progeny testing. We would continue this practice for future F_1 and F_2 progeny testing, meanwhile attempting to control extraneous variations introduced by inoculum quality or by variability in microclimates inside, or weather outside, inoculation tents.

2. Tony Squillace's 9-block experimental design had served well in the preliminary progeny testing, but had two failings. First, there were many seedlings lost from nongermination, damping off, and so forth, resulting in the number of seedlings in row-plots being substantially reduced; the binomial (percentage) data for row plots then became quite shaky. Second, the single row-plot per block provided no means for estimating within-block variance. We decided to increase the number of seedlings within a row-plot to 16, but balked at having two or more row-plots per block because of the consequent doubling of all operations and costs.

3. Continued reliance on controlled pollination would be safest for the near future. Meanwhile, we should experiment with various means for reducing pollination costs, first by using mixed-pollen crosses, and second by testing larger wind-pollinated progenies, possibly coming from mixtures of seed from 2 or more seed years.

4. Future R&D work undoubtedly would be more efficient and economical under some factorial or partial diallel crossing scheme. We decided that for future work of determining new GCA trees to use a factorial cross, each new and untested, rust-free parent being mated with four tester parents. This way we should be able to test many more parents than in the past for equal outlays in staff time and funds, and results between parents would be comparable, each new parent being represented by an equal number of test progenies including identical tester germ plasm.

5. We were expecting to encounter both elevation-associated variations (which we could handle in seed orchards as described above), as well as localized ecotypic variation (which with our 400-tree base we would be forced to ignore). Critical, long-range experiments were needed to confirm the extent of such variation and to prescribe the composition of seed orchards to handle the variation.

6. Inbreeding and associated depression of height growth, seed yield, and so forth was expected in *P. monticola* seed orchards. Critical experiments were needed to verify degree of inbreeding depression and to define possibly offsetting effects of selective fertilization. Orchard inbreeding coefficients should be calculated considering any selective fertilization effects.

7. Applied research was needed on *P. monticola* cone and seed yields, flower induction, vegetative propagation, and exploring other features of seed orchard technology.

8. In view of the high costs of empirical progeny testing, indirect selection for resistance—particularly seeking chemical and anatomical markers—should be explored.

The above assumptions, arbitrary decisions, and research recommendations shaped the new R&D program that followed. The preliminary research period indeed had been interesting and profitable.

A NEW R&D PROGRAM FOR THE 1960'S AND 1970'S

In 1954, the forest disease and insect control functions of the Bureau of Entomology, and similar research functions of the Bureau of Plant Industry, Soils, and Agricultural Engineering, were all finally blanketed into the Forest Service. This ended the crazy-quilt administration of forest pest control research. The Spokane Office of Blister Rust Control, BEPQ, then moved as a unit under a new Division of Blister Rust Control, Forest Service, Region 1, Missoula. This new division for a time retained control of all work of the D&I subunit. Financially, this probably was good because blister rust control work still was more amply funded than Forest Service research under the Experiment Station. Gradually, however, funding for new research in the Intermountain Station was increased to the point where in 1960 the Station took over administration of resistance research. By agreement, however, the Division of Blister Rust Control, and later the Division of State and Private Forestry of the Region, continued to finance any developmental work. This split the resistance R&D budget about 50–50 between the two agencies.

One of the bitterest pills I had to swallow as a member of the Forest Experiment Station was the decree that each research project would have detailed written problem analyses and study plans. Fortunately for me as a new research project leader, my immediate supervisor, Charles A. (Chuck) Wellner, chief of the Intermountain Station's Division of Forest Disease and Timber Management Research, was a close personal friend and a forest

researcher and research administrator of outstanding stature. Wellner was able alternatively to smooth my ruffled feathers and curb my tendencies toward empire-building. And, from 1960 to 1964, a detailed problem analysis and 17 study plans were prepared.

The problem analysis broke the overall research job into three major problem areas each with two to three phases, then cited specific studies aimed at solving the various phases. The major research problems and phases were:

I. Provision of resistant planting stock
   A. Early generation breeding
   B. Advanced generation breeding
   C. Seed orchard technology
   D. Supporting studies on inbreeding, elevational variation, and ecotypic variation

II. Increasing efficiency of selection
   A. Reducing time and cost of pollination work and progeny testing
   B. Indirect selection for anatomical and biochemical markers

III. Physiologic races of the rust
   A. Stockpiling additional resistance genes
   B. Genetics of the host:pathogen couplet
   C. Incorporating resistance genes from Eurasian white pines

Beyond this research program would be the developmental work of expanding the genetic base to 400 rust-free selections and about 100 GCA trees; also of any planning, preparing, and establishing the first phase seed orchards for mass production of resistant seed.

However, to illustrate the R&D program's balance of fundamental and applied research versus developmental work, the individual studies of the problem analysis are detailed below. Major Problem Areas (I,II,III) and Phases of the Areas (A,B,C,D), as covered above, are identified and study priorities are given. This is followed by a discussion of results that bore on the production of first-phase resistant planting stock.

Fundamental Research

QUANTITATIVE GENETICAL METHODS

I, B—highest priority: estimating first- to second-generation gain in resistance using quantitative genetical methods.

The steering committee and we scientists had decided that resistance substantially above 30 percent survival would be required to render planting stocks technologically or economically plantable. We also recognized that within our time frame, the increased resistance would have to come from one of two sources: (1) from substantially increased resistance of GCA-F_1 progenies in the field where subjected only to natural inoculation, presumably of much lower intensity but of much longer duration; or (2) from GCA-F_2 progenies that exhibited a substantial F_1 to F_2 gain in resistance under artificial inoculation. In either case, empirical determination of resistance seemed to be a 10- to 25-year proposition. Therefore, we decided to give first priority an attempt to **estimate** first- to second-generation gain in resistance from resistance data already in hand.

In the late 1950's, such heritability and genetic-gain analyses were new outgrowths in quantitative genetics (Lush 1956; Kempthorne 1957) and, except for the work of Toda (1958) and Toda and others (1959), were almost unknown in forest trees. Nevertheless, propelled by our urgent research needs, from 1957 to 1959 Tony Squillace and I plunged into the work of estimating second-generation gains in resistance under continued selection for GCA. Basic data for these analyses were percentages of survival determined for progenies of the 1952 test.

The review draft of the proposed research paper coming from this work was sent to frequent visitor and old friend Dr. Jonathan W. Wright, geneticist with the Department of Forestry of Michigan State University. It was returned so spattered with succinct commentary and so much improved by Wright's (then, to us, very sophisticated) suggestions for improved heritability analyses that we soon induced him to accept co-authorship of the paper. After review by a few quantitative geneticists, this first of our heritability and gain papers was published (Bingham and others 1960). Narrow-sense heritability was estimated as 0.688—an encouragingly high figure. We used the 30 percent gain in survival of GCA-F_1 progenies over the controls as the selection differential in the case of selection for GCA. The result was a genetic gain accruing to the second cycle of selection of $0.688 \times 0.30 = 0.21$, or 21 percent. Thus, combining first-(30 percent) and second-generation (21 percent) gains, it was estimated that second-generation GCA-F_2 progenies would contain about 51 percent seedlings capable of withstanding intense artificial exposure to the rust.

The estimated 50+ percent survival was probably the single most important figure we were ever to develop in the 25-year, first-phase, R&D program. This was because we researchers, our cooperators, and our steering committee all accepted that level of survival as adequate to justify large-scale planting of blister rust resistant planting stock. **This decision, in effect, locked us into a program for mass-producing GCA-F_2 stocks.** Immediately we commenced the expensive, 10-year program of developmental work test crossing and progeny testing the 330 new rust-free parents needed to bring the overall genetic base up to 400 trees and the base in GCA parents up to about 100 trees.

Less than a year later we were questioning the validity of some of our 1960 heritability analyses and recalculating first- to second- generation gain as perhaps only 10 percent. In spite of this, our cooperators and steering committee continued to give us the green light toward mass-production of the presumably 40 to 50 percent resistant GCA-F_2 planting stock. We made two later attempts to reestimate genetic gain (Bingham and others 1969; Becker and Marsden 1972). Based on four different progeny tests, estimated gains ran between 10 and 30 percent, estimated survival in the GCA-F_2's between 21 and 59 percent. Still later we found that the assumption of purely quantitative, polygenic inheritance of resistance probably was in error. Fortunately, by then we had highly encouraging empirical tests of resistance levels in GCA-F_2's, the results of which were available before we began installation of seed orchards. In other words, we "lucked out" on the matter of genetic gain; we might well have been producing F_2 planting stocks with a survival level under artificial inoculation that fell well below the acceptable but hypothetical 40 to 50 percent.

SEEKING CHEMICAL MARKERS

II, B—moderate priority: seeking chemical markers for possible use in indirect selection.

With costs for test crossing and progeny testing running at $1,000 to $2,000 per rust-free selection, we felt justified, even obligated, to investigate possible chemical and anatomical

markers for resistance; indirect selection using such markers well might reduce the high costs of selection.

In the fall 1958 Dr. James W. Hanover joined the Genetics Center in a new position specifically for investigating the chemistry of resistance. Over the next few years, Jim studied the relation of inorganic chemicals such as amino acids, organic acids, sugars, phenolics, and terpenes, to blister rust resistance in *P. monticola* (Hanover 1963a, b; 1966d; Hanover and Hoff 1966). This work was continued until 1969 by Dr. Raymond J. Hoff, who in 1964 assisted and then replaced Jim Hanover. Ray Hoff concentrated on a few of the more promising leads developed by him and Jim Hanover, notably with phenolics (Hanover and Hoff 1966; Hoff 1968; Hoff 1970) and with dry weight, where assisted by visiting German scientist Dr. Peter Schütt, (Schütt and Hoff 1969).

In the end, however, none of the potentially useful chemical markers proved consistently to be diagnostic of blister rust resistance. Nevertheless, we gained an extensive biochemical profile of *P. monticola* and, in time, some of the first knowledge on gene control of terpenes in *Pinus* (developed by Jim Hanover, later, while at Michigan State University; see Hanover 1966a, b, and c; and 1971). This was probably not too little to ask for our more than 5 scientist-man-years of work, especially considering the naivete of our approach. In retrospect, we realize the lack of success is not suprising now that we know just how few cells are involved in certain resistance reactions, or that we lacked genetic control, or even knowledge of some of the array of resistance genes.

THE PATHOGENICITY SYSTEMS

III, B—moderately high priority: genetics of the host: pathogen couplet.

By the mid–1960's, after failing to identify any chemical markers to resistance, we were ready to undertake new lines of research. We chose to study the genetics of the *P. monticola:C. ribicola* (host:pathogen) couplet because knowledge of the resistance and pathogenicity systems would be important for securing more stable resistance and because a wealth of new study materials was available.

In the process of developmental work increasing the program's genetic base from 70 up to 400 selections, over 300 new, rust-free, wild-stand selections were under test. Each new selection was represented by up to 160 seedlings in each of four test cross progenies, and at times (across several years of progeny tests) over 100,000 artificially inoculated seedlings were under test at one time. When rust examinations were to be made in the progeny test, we never seemed to have enough personnel, so all scientists, technicians, even secretaries, were blanketed into the inspection crews. This included Dr. Ray Hoff, and newly employed (1966) Dr. Geral I. McDonald. These two somehow managed to keep their heads above the waters of established examination routine far enough to make some astute observations on the host:pathogen couplet.

Ray Hoff and Geral McDonald first focused on the long-recognized but still unexplained needle-spots-only syndrome. Soon they established that the syndrome was a two-step resistance reaction: the first increment of resistance coming from premature shedding of infected needles, the second from the failure of the rust mycelium associated with certain remaining foliar infections to extend through the needle and short shoot (fascicle base) into the seedling's bark (McDonald and Hoff 1970). Then Ray Hoff pointed out the anatomical basis for the second increment of resistance, showing that the death of host cortical cells and of associated rust hyphae was occurring in and just distal of the host's short shoots (Hoff and McDonald 1971). Finally, the two scientists developed a statistically tenable genetic hypothesis accounting for the two resistance reactions and increments of resistance as found in the complete spots-only syndrome (McDonald and Hoff 1971). The hypothesis proposed a first recessive gene controlling premature shedding of infected needles, followed by a second recessive gene controlling failure of still established needle infections to spread through the short shoot and into the bark. This hypothesis remains unverified by other workers or with other materials; however, the materials Hoff and McDonald tested came from five different progeny tests, and in that sense the verification was repetitive.[10]

We never were able to develop a good explanation for the difference in the conclusions of quantitative (not major-gene) inheritance of resistance that Tony Squillace and I reached with the 1952 test versus that of major-gene inheritance that Hoff and McDonald reached. It seems highly unlikely that virulent races that had negated resistance of Hoff and McDonald's major-genes in the 1950's had since disappeared. One possible explanation is that the intensity of artificial inoculation somehow overrode the resistances found in the later tests; but this explanation also is unlikely because infection levels near 100 percent were reported by McDonald and Hoff (1970 and 1971). Still another explanation is that in the 1952 test effects of these major secondary foliage resistance genes were bypassed because the rust attacked via primary needles or directly via succulent bark tissues (Van Arsdel 1968), both as found on late-season lammas growth. This also seems unlikely because we have no records or recollection of much lammas growth with primary needles present, although we do have records of rare, infected primary foliage. Unfortunately, we ran out of large progeny tests and the opportunity to really verify the findings concerning these major resistance genes.

Evidence of other simply inherited forms of resistance was not long in coming. In spring of 1964, Ray Hoff and Geral McDonald observed and, using microscopic examination, verified that red as well as the common yellow needle spots were blister rust symptoms. Curiously, we had never noted such red spots, nor a reference or photo of them, prior to that time. But these red spots became a feature of every progeny test since undertaken. Within a few years, Ray Hoff and Geral McDonald had accumulated a large body of data on occurrence and frequency of the two colors of spots on a variety of test progenies. Thus, in time, proposing hypothetical genotypes for various parents, and checking these proposals via chi-square analyses of progenies, the two researchers were able to provide some of the first, fairly strong evidence for existence of pathogenic races in a forest tree rust (McDonald and Hoff 1975). The statistically tenable hypothesis involved one pathogenic race of the rust that produced yellow needle spot symptoms and faced off against a recessive resistance gene, and a second race that produced red needle spots and faced off against a dominant resistance gene; sort of a classic gene-for-gene system. This hypothesis also remains in need of confirmation. McDonald (1978) was unable to verify it after inoculating *P.*

[10]See appendix for anecdote.

monticola seedlings with sporidia from single spore aeciospore inoculated ribes leaves; the aeciospores came from clusters of aecia borne on single cankers on trees previously rated as red spotted only, red and yellow spotted, or yellow spotted only. However, this did not disprove the hypothesis because the mechanics of fertilization in *C. ribicola* remains to be clarified.

Ray Hoff and Geral McDonald (1972b) went on to summarize the several resistance reactions and hypotheses concerning their control by resistance genes as follows:

1. Resistance to yellow-spot-forming race (single recessive gene), to red-spot-forming race (single dominant gene), and, by inference, to both races (that is, no spots).
2. Reduced lesion frequency on secondary needles (single nondominant gene or gene(s) of uniform resistance type).
3. Premature shedding of infected needles (single recessive gene).
4. Fungicidal reaction in the vicinity of the short shoot (single recessive gene).
5. Rapid necrosis of bark surrounding infected needle bundle bases (sort of an overblown bark hypersensitivity reaction-genetic control unknown).
6. Corking-out of established bark cankers (extensive wound-periderm formation, genetic control unknown).
7. Slow canker growth (genetic control unknown but probably of uniform resistance type).

Ray Hoff and Geral McDonald pointed out that while most forms of resistance found probably were of the vertical (or differential) kind (single, major-gene-controlled, and thus requiring but a single mutation by the rust for negation), at least the reduced needle lesion frequency and slow canker growth forms of resistance appeared to be of the more stable horizontal (or uniform) or tolerance types. However, they also pointed out that certain vertical types of resistance, such as spots-only resistance, had persisted for long periods in species such as *Pinus griffithii* and *Pinus armandii* (Hoff and McDonald 1972a,b) from near the central Asian *C. ribicola* gene center.

Basically then, this was the resistance information available for planning the developmental work toward seed orchard production of first-phase resistant stock.

ELEVATIONAL AND ECOTYPIC VARIATION

I, D—high priority: effects of elevational and ecotypic variation.

The Inland Empire's natural stands of *P. monticola* in the U.S.A. extend across a scant 3° of latitude or longitude; thus, we were not anticipating much geographic variation in the species. The elevational range of the species, however, is more than 3,000 ft (915 m), so we were expecting some elevation-associated genetic variation. This proved to be the case. Our earliest results on height growth in *P. monticola* (Squillace and Bingham 1958a) showed high-elevation lots, from one 5,000 ft (1 525 m) area exhibiting slow growth at age 2 years in a low-elevation nursery, but fairly good growth on a 4,400 ft (1 340 m) outplanting plot at age 4 years. Furthermore, the same study indicated that *P. monticola* contained localized, site-associated variation. Thus it was that Dr. Burton V. Barnes (who replaced Tony Squillace in 1958) immediately commenced study of the elevational and other variations over the entire range of Inland Empire *P. monticola*. By 1967 Barnes had shown that in one long northern Idaho drainage, the **phenotypic** variation in periodic annual height growth differed significantly only at the highest elevations, over 4,600 ft (1 400 m) (Barnes 1967).

We recognized that this natural genetic variation was a possible stumbling block to securing adaptation of resistant planting stock. We also recognized that critical studies confirming the extent and importance of elevational and localized ecotypic variation, while of high priority, were long-range, and that we would still be awaiting answers when it came time for the first-phase orchards to be established. Thus, lacking firm answers, we merely set up low-, medium-, and high-elevational zones for all GCA trees and corresponding low-, mid-, or high-elevational seed orchards.

Unfortunately, seed orchards for the production of low-, mid-, and high-elevation, resistant F_2 stocks already had been established and growing for 5 years before we could obtain more definitive information on elevational and localized ecotypic variation. First Dr. Raphael J. (Ray) Steinhoff (who replaced Burt Barnes in 1965) interpreted results on tree growth for up to 16 years from 4 nursery tests and 13 field plantations. He showed conclusively the lack of elevationally associated variation in growth except in *P. monticola* materials from the highest elevations of over 4,500 ft (1 375 m) (Steinhoff 1979). Second, Dr. Gerald E. (Jerry) Rehfeldt (the project's "gene-ecologist" who arrived in 1967)—working with control-pollinated progenies from 3,100, 3,850, and 4,600 ft (950, 1 175, and 1 400 m) showed that Squillace and Bingham's (1958a) localized ecotypic variation probably was a myth—an artifact of the particular materials studied or of experimental error. Instead, Rehfeldt (1979) found little variation associated with aspect or elevation, except at the highest elevations. He also pointed out how such "phenotypic plasticity" could well represent an alternative adaptive strategy to the relatively complex patterns of populational differentiation we were finding in other Inland Empire conifers. Rehfeldt (1979) also cited other studies on height growth and terpenes of *P. monticola* that supported the "phenotypic plasticity" hypothesis for the species (Hunt and von Rudloff 1977; Townsend and others 1972).

Long-range payoffs from detection of this plasticity in Inland Empire *P. monticola* are self-evident. Seed from currently established low- and mid-elevation orchards can be lumped and planted over a much wider range of elevations than had been anticipated. Also, this plasticity will greatly simplify the structuring of future seed orchards, in effect increasing the genetic base of materials entering a given orchard (Hoff and McDonald 1980a).

SELFING AND SELECTIVE FERTILIZATION

I, C—moderate priority: effects of selfing and selective fertilization on *P. monticola*

Bingham and Squillace (1955) showed that under controlled self- pollination of *P. monticola*, the bulk of individual trees proved to be partially self-fertile, and that the selfing was accompanied by an almost 60 percent drop in number of seedlings produced and a more than 20 percent reduction in height growth of young seedlings. Soon we would be considering grafted seed orchards with many genetically identical ramets of each GCA tree ortet, or resistant F_1 seedling seed orchards, with many full sib seedlings of each GCA-F_1 progeny. It behooved us, then, to know more about effects of selfing in older trees, and about possibly offsetting effects of selective fertilization under wind-pollination of seed orchard trees.

By 1964 we knew that inbreeding depression of height growth in S_1 progenies persisted through age 10 years, and appeared to have increased to near the 30 to 40 percent level (Barnes 1964). But we also knew that there were strong selec-

tive fertilization effects favoring outcross pollens in mixes of self and outcross pollens. In fact, some completely self-fertile trees might be mostly outcrossed depending on the pollinators. And with certain partially self-fertile trees, outcross pollen might be as much as five times as effective as self pollen in effecting fertilization (Squillace and Bingham 1958b; Barnes, Bingham, and Squillace 1962).

On reflection, however, our concern about the effects of inbreeding in seed orchards was probably "much ado about nothing." The Sandpoint experimental grafted orchard (see "Seed Orchard Technology") was composed of 13 clones and had a potential inbreeding coefficient of 0.077; the final first-phase seedling orchards would be composed of 12 full-sib lines with a potential inbreeding coefficient of 0.010. Thus, if there were completely panmictic fertilizations in these two orchards and 35 percent inbreeding depression of height growth under full inbreeding, the corresponding depressions of height growth should amount to only about 2.7 percent in trees from Sandpoint and less than 0.5 percent in trees from first-phase orchards.

We worried even less about effects of inbreeding in our seed orchards when we added the following facts: (1) any inbreeding probably would be accompanied by a decrease in seedling yield; (2) outcross pollens probably would be favored in effecting fertilization; and (3) that ramets of the 13 Sandpoint ortets, or half-sibs of the 12 full-sib lines of the first-phase orchards, were or would be physically separated by using a spacing system such as that of Langner (1953).

Applied Research

F_1 TO F_2 GENETIC GAIN

I, B—high priority: empirical determination of F_1 to F_2 gain under artificial inoculation in the nursery.

After 1960–61, with the estimate of more than 10 to 20 percent gain in seedling survival between the F_1 and F_2 generations, and 40 to 50 percent or more survival in GCA-F_2 progenies (Bingham and others 1960, 1961), our developmental work program was almost locked onto F_2 seedling seed orchards. Thus, verification of the actual F_1 to F_2 genetic gain became a high priority study, especially because results would probably be available in time to forestall installation of seed orchards should the 1960 and 1961 estimates of gain prove to be unrealistically high.

Beginning in 1957, resistant GCA-F_1 seedlings from the preliminary 1952 to 1955 progeny tests were salvaged from field plots and accumulated in the fertile, sprinkler-irrigated, fertilized, and cultivated Moscow Breeding Arboretum. The oldest of these resistant F_1 seedlings had begun to produce female strobili by age 7 (in 1958) and male strobili by age 12 (in 1963). Significant production of female strobili occurred at age 10 (in 1961) and of male strobili at age 14 (in 1965). By 1967 we were able to enter a fair number (32) of F_2 progenies into the regular progeny tests used to determine new GCA trees.

The outcome of these tests involving F_2 progenies was far more encouraging than we had expected based on previous experimental estimations of genetic gain (from estimates of Bingham and others 1960, 1961, 1969, Becker and Marsden 1972). Instead of 50 percent or less of the GCA-F_2 seedlings surviving artificial inoculation, we found that more than 65 percent survived (Hoff and others 1973).

Apparently the increase over estimated percentage of survival was due to the involvement of major genes in the resistance system. However, we were unable to substantiate this hypothesis because certain F_1 parents we thought were homozygous recessive for one of the spots-only syndrome resistance genes, produced F_2 progenies with only 88 percent survival. Possible explanations for the less than 100 percent survival in some F_2 progenies were that infections had occurred via primary needles on lammas growth or persistent from the year previous to inoculation or directly via succulent bark (Van Arsdel 1968). However, in the latter case, after 20 years and more of progeny testing of more than 250,000 inoculated seedlings, such direct stem penetration has never been demonstrated here for *P. monticola*.

These findings, first known in late 1970, provided the final impetus for proceeding toward GCA-F_2 seedling seed orchards.

LEVELS OF FIELD RESISTANCE

I, A and B—high priority: empirical determination of levels of field resistance in F_1 and F_2 stocks.

Until 1970, as described for the foregoing study, we had only estimates of the level of resistance that would be attained in artificially inoculated F_2 stocks, and no knowledge that field resistance, even in F_1 stock, might not be great enough for practical use.

Some preliminary results on field resistance of GCA-F_1 progenies was obtained by Ray Steinhoff (1971) on 16 progenies Tony Squillace had planted from 1955 to 1959. These progenies had been exposed only to natural inoculation for 12 to 16 years at Priest River and Deception Creek Experimental Forests. The GCA-F_1 stock on the two field plots showed 18.5 to 20.9 percent of the seedlings infected. Concurrently, controls were 48.4 to 68.0 percent infected, and natural reproduction was 62.5 to 80.1 percent infected.

Additional information on field resistance of both GCA-F_1 and GCA-F_2 stocks was obtained in 1973 from a large planting specifically designed to provide information on field resistance. Here, on an extremely high- hazard site, after 3 years of field exposure and with 2 years of rust infection visible, GCA-F_1 stock was 31 percent infected, GCA-F_2 stock 12 percent infected, and controls 76 percent infected (Bingham and others 1973). After 7 years of exposure and 6 years of visible rust, F_1 stock was 47 percent infected and 9 percent dead due to rust, F_2 stock 27 percent infected and 4 percent dead, and controls 92 percent infected and 27 percent dead.

Our latest and longest duration information now comes from Tony Squillace's 1955–59 outplantings. Visiting scientist Dr. Ray E. Goddard from the University of Florida and Geral McDonald reexamined Tony Squillace's plots in summer 1980. Now 21 to 26 years after first exposure to natural inoculation, GCA-F_1 stock was 31 to 48 percent infected; controls, 69 to 86 percent infected; and natural reproduction 86 to 99 percent infected, on three outplanting plots.

It begins to appear, particularly in low-rust-hazard areas, that the level of field resistance will indeed be higher than that determined experimentally under artificial inoculation.

IMPROVEMENTS IN PROGENY TESTING PROCEDURES

II, B—moderate priority: reducing the time and cost while increasing the accuracy and sensitivity of pollination and progeny testing work.

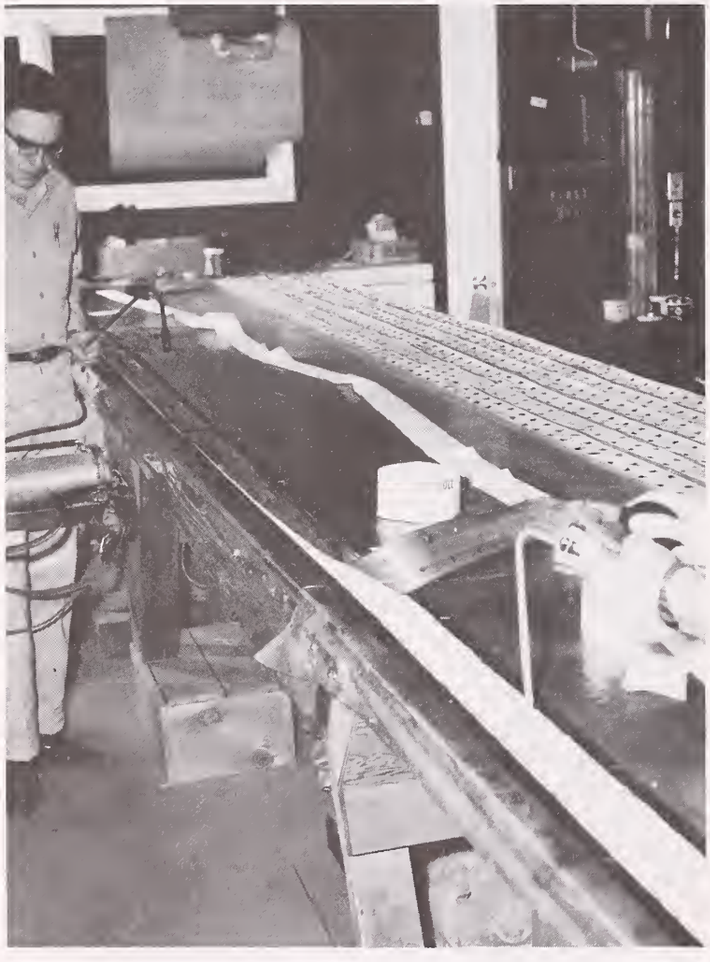
As already mentioned, the newer progeny tests of the 1960's were made using series of four heavily fruiting GCA parents as testers in a 4-tester factorial cross, as well as a 10-randomized block design with 16 seedlings per test progeny row-plot. This pollination and test procedure proved to be considerably less expensive for detecting new GCA trees and more sensitive for heritability analyses of resistance (Bingham and others 1969; Becker and Marsden 1972; Hoff and McDonald 1980b) or for heritability analyses of height growth (Hanover and Barnes 1963; 1969).

By 1964 seed were presown on stenciled, 10-block long, biodegradable paper strips immediately after extraction in the greenhouse, and up to 75,000-seedling progeny tests were fall-sown in the nursery in predivided and tagged nursery beds in the course of a single day (fig. 13). Thus, we had abandoned seed stratification and merely overwintered seed in the nursery beds.

We were also trying to remove controllable variation from the artificial inoculation process. We sheltered the ends and edges of seedling beds with water-soaked burlap during inoculations, and we tried to even out inoculum differences inherent in leaves from different Ribes spp. plants by detaching and mixing up the leaves as they were spread out on nursery bed screen-covers placed above the pine seedlings. We met with only limited success, with inoculation intensity across the block, even within the same block, still varying widely (fig. 9). (See also Patton [1972] and Bingham [1972] for unsolved problems with degree and uniformity of artificial inoculations.)

One of our more common-sense improvements came from Hoff and McDonald's studies of the spots-only syndrome. On their recommendation, we moved the time for rust examination from summer and fall to spring, usually in June. This greatly improved our capabilities for detecting shedding of spotted needles.

We also tested the reliability of single, mixed-pollen crosses for reflecting average performance of selections as based on the 4 tester crosses of the standard factorial cross. Based on percentage of infection, crosses that were made with equal volume mixtures of the pollens of the 4 testers or with those pollens plus pollens of 6 other trees (10-tree mixes), reflected the average tester cross quite closely. One year mixed-pollen crosses underestimated percent of infection determined from the average tester cross, while a second year the mixed-pollen crosses overestimated percent of infection. The 10-pollen mixes generally were more reliable in that percentages of infection deviated less from the average of the 4 tester matings (Bingham 1967, 1968). The mixed-pollen crosses, however, never were used in our first-phase progeny testing program. Results came rather late. Also, they were less sensitive crosses for heritability or other analyses we wished to make.



(A)

(B)

Figure 13.—How we handled the really large progeny test seed sowing jobs after 1960. (A) Seed spots were stenciled onto a strip of biodegradeable toweling using a sprayed dye solution (left). Then a biodegradeable methyl cellulose adhesive was dropped on each stenciled seed spot and the desired number of seed dropped thereon. The 10-block-long strip of toweling was then dried (at rear on table), cut apart at block lines, and stored till sown outdoors. (B) Seed on paper-towel strips were sown in the Moscow, Idaho, blister rust nursery in the fall and covered with sand. When germinated they were thinned so that there was one seedling per planting spot, or 16 seedlings for each test progeny per randomized block.

SEED ORCHARD TECHNOLOGY

I, C—moderate priority: studies in *P. monticola* seed orchard technology.

From 1960 on, with good levels of resistance in store, seed orchards looked more possible. We began investigating vegetative propagation, cone and seed yields in seedling and grafted trees, and other matters of P. monticola seed orchard technology that would set the character and size of future seed orchards.

We established that average cone and seed yields for young P. monticola trees in nature were 28 cones with 104 filled seed per cone, or 2,900 seed/tree/year (Bingham and Rehfeldt 1970). Then, using the same 18-year records, we analyzed the factors affecting periodicity of yield in nature (Rehfeldt and others 1971). We also established the extent of insect-caused cone and seed losses in *P. monticola* and showed that in certain areas and seed years, cone beetles (*Conophthorus ponderosae* Hopkins) destroyed 90 percent of the cones, while cone moths (*Eucosma recissoriana* Heinrich and *Dioryctria abietivorella* [Grote]) attacked and partially destroyed 50 to 85 percent of the cones (Barnes, Bingham, and Schenk 1962). Even the isolation of *P. monticola* (as at Moscow, Idaho, about 10 miles from the nearest natural stands of the species) failed to eliminate the cone moth, and we wonder when the cone beetle will enter the scene there. Seed orchard insect controls still remain to be developed.

Barnes and Bingham (1963a and b), on plots installed by Tony Squillace, also investigated top-grafting of young scions into large and reproductive mature trees, as well as 5-year effects of field fertilization, cultivations, and irrigation for "inducing" strobilus development on P. monticola seedlings 6 to 11 years old. None of these field treatments seemed to have much effect in hastening or increasing strobilus bearing in the young trees. But the three cultural treatments, alone or together in any combination, definitely affected growth. Meanwhile at the Moscow breeding arboretum—under a regime of sprinkler irrigation that added 10 to 15 inches (25 to 40 cm) of diluted sewage effluent, along with clean cultivation—more than 16 percent of 11-year- old P. monticola trees bore female strobili.

We also have had opportunities to study clonal variations and effects of graftage on cone and seed production, basing our observations on the 17-acre grafted seed orchard established mainly by Jim Hanover at Sandpoint, Idaho, in 1960 (Bingham and others 1963). First, Hanover (1962) showed that individual ortets varied in graftability, and that through 20 months success in grafting was apparently associated with vigor of ortet-shoots used for grafting. Then we noticed that 6 of 13 of the ortets were to some extent incompatible with the nursery- run, Kaniksu National Forest rootstocks. Incompatibility was delayed for up to 13 or more years (Hoff 1977). Later Ray Hoff and Geral McDonald (1978) demonstrated a highly significant difference among the ramets of the 13 ortets in intensity of infection by a needle blight disease associated with a *Lecanostricta* species. Despite trouble with scion-stock incompatabilities, cone and seed production at the Sandpoint grafted seed orchard has been spectacular. In 1980, an otherwise good cone year, and 20 years after orchard establishment, many grafted trees were producing a bushel of cones and a half-pound (225 gm) of seed apiece. This same year, the older (25 to 29 years) F_1 trees of the Moscow Breeding Arboretum were producing only about 31 cones and 1,209 filled seeds per tree. And at Moscow there still seemed to be a pollination problem, as witnessed by the very low yield of 39 filled seed per cone (Hoff, personal communication).

Developmental Work

ESTABLISHING SEED ORCHARDS

Our program of fundamental and applied research of the 1960's provided most of the answers we needed to determine the genetic structure, kind, size, and location of seed orchards for production of blister rust resistant P*P. monticola* planting stock for the Inland Empire. The only important information still lacking was on the importance of elevation-associated variation in *P. monticola* and on the long-term field resistance of the selected GCA-F_1 and GCA-F_2 progenies. By about 1968 we were ready to plan the structure and establishment of seed orchards.

First, however, the Forest Service units cooperating in the R&D program had to make some basic decisions and assumptions about the character and size of the first-phase seed orchards:

1. We would produce only GCA-F_2 seed in seed orchards composed of resistant GCA-F_1 seedlings.
2. The genetic base of the orchards would be pegged at about 100 GCA trees we had found in the 400 rust-free selection base, but more stability of resistance would be sought by selecting for a variety of resistance reactions (and presumably, resistance genes) in the individual GCA-F_1 seedling orchard foundation stocks.
3. There appeared to be significant elevation-associated variation in *P. monticola*. We assumed that an arbitrary division of selections, planting sites, and seed orchards among low- (below 3,500 ft, or 1 065 m), mid- (over 3,500 to 4,100 ft or 1 066 to 1 250 m), and high-elevation (over 4,100 ft) zones would be used to control maladaptation of seed orchard planting stocks. These three zones were estimated to comprise about 32.5, 50, and 17.5 percent, respectively, of the 2 million acres of Inland Empire white pine lands.
4. The size of the seed orchards would be determined by the following considerations:

   a. Trees would be spaced at 20 ft by 20 ft (6 m by 6 m) in orchards, and reserve stock would be maintained by planting two foundation stocks at each planting spot.

   b. For the time being we would plant resistant stocks only in the better 1 million acres of white pine lands where (as rotation age is set between 50 and 100 years) 1 to 2 percent (or 10,000 to 20,000 acres) of these lands would become available for planting annually—through clearcutting, underplanting of shelterwood cuts, or wildfires.

   c. The conservative assumption—that field resistance would not exceed the 65 percent found experimentally under artificial inoculation—would hold, and also assuming tubed planting stock and high planting survival, then the desired stocking of about 200 fairly evenly spaced trees per acre (at about 15 ft by 15 ft spacing; i.e., 500 trees per ha at 4.5 m by 4.5 m) would be attained by allowing for 35 percent rust losses and planting at about 300 trees per acre (at about 12 ft by 12 ft spacing; i.e., 740 trees per ha at 3.6 m by 3.6 m).

   d. Under routine nursery practice, there would be a 50 percent loss between numbers of filled seed and numbers of plantable seedlings.

5. Orchards would be situated where isolated from natural white pine stands and where seed production would be favored by relatively long, high-temperature growing seasons and the application of irrigation water.

6. These first-phase seed orchards, once in production, probably should not be used for longer than 20 years—or beyond the date when broader based materials of sufficient field resistance become available.

These assorted decisions and assumptions largely set the size, type (seedling), and general locality of the three seed orchards, and to some extent, established their genetic structuring.

SIZE AND LOCALITY OF SEED ORCHARDS

The specified planting of resistant F_2 stocks at 12 ft by 12 ft (3.6 m by 3.6 m) spacing would require 302.5 trees per acre, so that the total number of seedlings required to plant 20,000 acres per year would be 6,050,000. Using the figure of Bingham and Rehfeldt (1970) of 2,900 seed per young *P. monticola* tree per year (probably conservative because figures come from uncultured, natural-stand trees), the 109 trees spaced 20 ft by 20 ft (6 m by 6 m) on each acre of seed orchard would produce 316,100 filled seeds (781,000 per ha). After 50 percent are lost in the nursery, about 158,050 plantable seedlings remain (390,550 per ha). Thus, for an annual production of 6,050,000 plantable seedlings, almost 40 acres (16 ha) of seed orchards would be required.

Fortunately, the problem of locating and securing lands for the seed orchard was quickly solved. Through the foresight of former Coeur d'Alene National Forest Supervisor Ray Hilding, a quarter-section (160 acres; 65 ha) of relatively flat, marginal-agricultural lands with *Pinus ponderosa* Laws. and *P. contorta* Loud. stands had been held despite its demonstrated value for lands trading and consolidation. It was 5 miles (8 km) or more distant from natural white pine stands, had a relatively long growing season, and probably would be underlain by aquifers adequate for its sprinkler irrigation. Thus it was that 27 acres (11 ha) of this quarter-section, located near Lone Mountain on the Rathdrum Prairie northwest of Coeur d'Alene, Idaho, were to be dedicated to *P. monticola* high- and mid-elevation seed orchards. The area since has become a center for Forest Service Region 1tree breeding work (fig. 14). Another 13-acre (5-ha) low-elevation orchard was located on otherwise useless hilly terrain along the south border of the Forest Service's Coeur d'Alene Nursery.

Figure 14.—The so-called low elevation *P. monticola* F_2 seed orchard at Lone Mountain northwest of Coeur d-Alene, Idaho.

FOUNDATION STOCKS

The 40 acres (16.2 ha) of *P. monticola* seed orchards, each double planted at the 109 planting spots per acre, would require a total of 8,720 of the resistant, GCA-F_1 foundation stocks. This number would consist of 1,526 high-elevation plants for 7 acres (2.8 ha) of high-elevation orchard; 2,834 low-elevation plants for 13 acres (5.3 ha); and 4,360 mid-elevation plants for 20 acres (8.1 ha). Thus, we would require a total of 128, 236, and 364 foundation stocks, respectively, from each of the 12 high-, low-, and mid-elevation GCA-F_1 families (see below).

Aside from the joint decision of cooperators to include a variety of resistance reactions (and probably resistance-genes) in the three elevational seed orchards, the actual structuring of resistance therein was mostly left to us researchers. Here's how we attempted to structure resistance.

The 1952 to 1967 progeny tests had tested 400 rust-free parent selections and uncovered about 100 GCA trees among them. Naturally, there were not exactly 33.33 GCA trees found for each of the three arbitrary elevational zones. In fact, we found only 24 GCA trees for one zone and then decided to use only the best 24 GCA trees as foundation stocks for each of the orchards. Thus, only the 72 best of 100 GCA trees were entered in the three first-phase seed orchards.

Then, between 1965 and 1968, after the best GCA trees for each zone had been identified, we commenced mating the 24 GCA trees for each zone in a series of 12, very large, unrelated, F_1 matings. These matings involved sometimes more than 50 pollination bags and ultimately produced more than 3,000 seed or 1,500 seedlings. The resulting 36 GCA-F_1 seed lots were sown (about 8 to 10 lots each year) between 1967 and 1970. Then they were artificially inoculated at 2 years of age. At 4 years of age the correct numbers (128 to 364) of resistant GCA-F_1 seedlings (that is, the seed orchard foundation stocks) were selected and tagged from each of the 36 GCA-F_1 progenies. Finally, in the early spring of their fifth growing season, 1971–74, the tagged seedlings representing the 12 pertinent GCA-F_1 families were outplanted into the three elevational-zone orchards.

Actual structuring of resistance was then accomplished as follows: One-third of the resistant GCA-F_1 foundation stocks in each of the 12 GCA-F_1 families, in each of the three orchards, was chosen to represent the premature-needle-shedding resistance reaction (and its presumably recessive, associated gene) of the spots-only syndrome. A second one-third was chosen to represent the short shoot fungicidal resistance reaction (and its presumably recessive, associated gene) of the spots-only syndrome. A final one-third was chosen to represent various bark reaction resistance types.

Knowing what we do today about structure of resistance in GCA trees, this artificial selection scheme promises to provide fairly stable resistance. Resistance in the presumably 65+ percent surviving GCA-F_2 seedlings coming from these seed orchards should be based on at least five (three vertical and two horizontal) types of resistance reactions. The two horizontal (or uniform) types of resistance are involved because in selecting for GCA about half of the parents also exhibit low needle lesion frequency, and 30 percent of the parents show slow canker growth.

SUMMARY

Twenty-five years of research and development work (1950–75)—first-phase work undertaken by Forest Service cooperators—has led to experimental production (and soon mass-production) of Inland Empire western white pines bred for blister rust resistance. Breeding has gone through two generations, until 65 percent of the trees resist intense, artificial exposure to the rust fungus. And unless the racial structure of the rust alters disastrously, the long-range survival of these second generation stocks under natural exposure to the rust probably will exceed 65 percent.

Resistance in the second generation stocks is based on selections for general combining ability for a combination of differential and uniform types of resistance. Some of the resistance reactions—and, presumably resistance genes—are identical to those that probably have persisted for long periods near the Asiatic white pine:blister rust gene center. Thus, resistance in these first-phase stocks will probably persist until we can produce faster growing and better adapted second-phase stocks embodying more types of resistance genes and more stable resistance.

If this R&D program has been a "success story," then it's mainly because the biological, research, and administrative climates were all ideal. R&D workers had only to rely on a large backlog of information on disease resistance in agronomic crops and to interpret correctly experiences with resistance in eastern white pine and Eurasian white pines to attain an almost certain success.

WHERE DO WE GO FROM HERE? PHASE II

As stated at the beginning of this report: "Implied in the term 'first-phase' was the idea that planting stocks would continue to be improved in subsequent programs—toward successively faster growing, better adapted, and more resistant stocks embodying more resistance genes and more stable resistance." What sort of progress have we made toward these goals in the first of these subsequent programs, labeled the "second-phase" program?

Phase II work commenced in 1967 under the Intermountain Forest and Range Experiment Station, the Northern Region, and the several "white pine" National Forests, using Congressionally appropriated, Forest Service blister rust control funds. The progeny testing part of the work is continuing today under an eight-member, cooperative, Western White Pine Tree Improvement Committee within the parent Inland Empire Tree Improvement Cooperative. Current members are the Forest Service, Region 1; the University of Idaho; the Department of Lands, State of Idaho; the Coeur d'Alene Tribe, BIA; Diamond International Corporation; Idaho Pines Timber Associates; Potlatch Corporation; and St. Regis Paper Company. Other private industry cooperators are considering membership.

In 1967, the first job undertaken by the white pine Forests was to expand the phase I genetic base of rust-free, wild-stand, "candidate trees" from 400 up to 3,200 (400 phase I and 2,800 phase II). We came within about 100 trees of attaining the 2,800-tree phase II goal—actually locating 2,698 new trees. With the 400 phase I trees, we then had 3,098 trees in hand, of which 161 later were dropped because of accidental destruction, death by disease or insects, having too many blister rust cankers, or other reasons. At present, the remaining 2,937 trees can be divided into two potential breeding populations, as follows:

Potential breeding population	Number phase II candidates including phase I trees
Northern Idaho-Northwestern Montana white pine lands less than 4,500 ft (1 370 m)	2,533
Northern Idaho-Northwestern Montana white pine lands greater than 4,500 ft (1 370 m)	404
Total number phase II candidates	2,937

Originally this 2,937-tree genetic base was to have been compartmented latitudinally and elevationally, and possibly longitudinally if found necessary according to the performance in Squillace, Barnes, or Steinhoff provenance tests. Fortunately, however, Rehfeldt (1979) and Steinhoff (1979) have demonstrated the remarkable phenotypic plasticity of *P. monticola*, removing the necessity for compartmentalizing the base except, perhaps, to remove the 404 high-altitude candidates.

It was soon apparent that little in the way of meeting the phase II program goals could be realized by breeding within the relatively small, 404-tree, high-elevation population. In fact, it appears that excluding these 404 trees from the overall phase II base population would merely be gilding the lily. The following supports this view:

1. Elevation accounts for only about 2 percent of the variation in height growth in *P. monticola* (Steinhoff 1979).
2. Even though height growth was reduced about 10 percent in progenies of high-elevation *P. monticola* trees (Steinhoff 1979), if the 404 high-elevation trees were added to the 2,533-tree low- and mid-elevation phase II population, the loss would be diluted to less than 2 percent.
3. If the low-, mid-, and high-elevation trees were all included in the same breeding population, then, barring cold injuries, we might expect an offsetting increase in height growth when seed orchard planting stocks were used on high-elevation sites.
4. Even a low-intensity family selection for fast height growth would eliminate more of the high- than the low- or mid-elevation trees.
5. Lastly, Rehfeldt's latest (1982) information shows the height differences associated with the highest elevations to be nonsignificant.

At the same time there is ample opportunity for improving the 2,533-tree low- and mid-elevation population, or the overall 2,937-tree population. Selection will take place at the conclusion of 6 years of progeny testing, using 180-tree, wind-pollinated progenies. Utility and reliability of wind-pollinated progenies for appraising resistance have increased markedly over the years as the proportion of nonresistant trees in the residual populations has been reduced (Hoff and others 1973). Selection priorities we have tentatively assigned are as follows:

First priority – Family, then individual tree selection for three uniform (horizontal) resistance types (that is, low needle-spotting frequency, slow appearance of bark cankers, and slow growth or tolerance of bark cankers).

Second priority – Individual tree selection, first for any uniform resistance types not selected above, then to four differential (vertical) resistance types including complete lack of foliar infection, premature shedding of spotted needles, short shoot fungicidal reaction, and various bark resistance reactions.

Third priority – A low-intensity, family selection for rapid, early growth rate.

Some simple arithmetic shows that if we are to meet primary phase II objectives of increasing the kinds and stability of resistance (as under first and second priority selections, above), then there will be only limited possibilities for improvement of growth rate. For instance, suppose in the 2,937-tree, total phase II base population, we are forced to drop 437 more trees because of limited cone and seed bearing, poor seed germination in progeny tests, other progeny test "accidents," extreme susceptibility of progenies, or other reasons. Then suppose we select the upper 50 percent of the remaining 2,500 trees in a family selection for low needle spot frequency. Then suppose we select the upper 20 percent of the 1,250 remaining trees in a family selection for slow canker growth rate, further reducing the base to 250 trees. Continued family selection for the third uniform trait probably would be unwise, reducing the base to less than 100 trees; so individual tree selection for the remaining uniform and the four differential traits would have to be instituted. Under these conditions, there is small promise for making family selections for growth rate for other than the perhaps upper 25 percent of the 250 trees.

The 2,937 phase II candidate trees are being progeny tested in five testing cycles running from 1976 to 1989 at the Coeur d'Alene Forest Service Nursery. Various uniform and differential resistance reactions are being identified and marked with varicolored plastic rings on individual seedlings, awaiting final family and individual progeny tree selections. Seedling seed orchards should be established between 1982 and 1989 and should bear seed by significant amounts of about 2000.

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APPENDIX

This appendix contains the material for footnotes 2 through 10 in the text. Because of the lengthy and anecdotal nature of these footnotes, they have been kept here as a separate section.

[2]An interesting sidelight on researchers developed about this time—in the 1930's. German blister rust researcher and authority, Professor C. F. von Tubeuf, had been waging somewhat of a crusade against various German forest administrators who persisted in advocating the planting of susceptible North American white pines in German State Forests (von Tubeuf 1905, 1917, 1924, 1928, 1936). Somehow von Tubeuf came across his countryman Professor J. Liese's remarks that the interracial resistance to *Lophodermium pinastri* in *P. sylvestris*, indeed might hold for *C. ribicola* resistance in *P. strobus*. Von Tubeuf's (1935) reaction, in the manner then popular in the very lively German forestry periodicals, was to publicly scold Liese, saying (the author's translation), "Liese's beliefs in relation to immune *P. strobus*, and that resistant varieties can be produced through breeding, does not help us now" (apparently in saving infected *P. strobus* stands or preventing further introductions). "Has he discussed this with Professor Dengler?" (apparently A. Dengler, a prominent German silviculturist of the time, who must have agreed with von Tubeuf). "Can he lay out for us one practical breeding plan? Has he undertaken research in this area that is favorable? I think not'!" Apparently this harangue did not much deter Liese. Later, Liese (1936) said that *P. strobus* well might display racial variation in resistance to *C. ribicola*, simply referring von Tubeuf to his (Liese's) rather definitive experiments establishing the racial variation of Scotch pine resistance to *P. pini* (1930a,b, 1936) where again he extrapolated possibly similar results in resistance of *P. strobus* to *C. ribicola*.

[3]Much of this is hearsay, but probably it's worth preserving.—The annual, summer 1949 field trip of the Idaho State Land Board (the agency that administers the education-supporting funds coming from timber cut on State lands) was under way, very grandly transported by river-drive, wanigan-raft, down the very remote and beautiful North Fork of the Clearwater River in northern Idaho. One main purpose of this particular trip was for Land Board members and their blister rust control administrator guests to consider the acceleration of timber harvesting plans in the State-owned, heavily rusted mature *P. monticola* stands that bordered the river. One exchange between a Land Board member and a now deceased but then leading blister rust control administrator was leaked to the author about as follows: Land Board Member—"I understand that University of Wisconsin researchers are already at work exploring blister rust resistance in *P. strobus*; are your people planning anything along these lines in *P. monticola*?" My informant tells me there was a pregnant pause, and then, as if suddenly remembering the lonely box of cuttings protruding from the 8th floor window in Spokane, or the single, controlled pollination attempt, the blister rust control administrator finally answered, "By golly, we're already working on that!" Perhaps this hearsay deserves some credence, for I can testify that the administrator did, a few weeks later, visit the 8th floor lab and did, as usual, casually glance at the windowbox cuttings, and then did ask me pointblank, "Shouldn't we be doing something more toward development of blister rust resistance in *P. monticola*?" Fortunately, he did not inquire into the abortive pollination. (The author has since publicly confessed that he had pollination-bagged the previous year's [but still small] cones, and later, after the pinkish and strangely different current-season strobili began to emerge from the branch-tip buds, surreptitiously moved the pollination bags where they belonged.) As Tony Squillace points out in his review of this paper, it is significant that I soon was sent to the Placerville, Calif., Institute of Forest Genetics to learn how to breed trees.

[4]It was only much later that we had sophisticated apparatus like a cone-tumbling drum to shake winged seeds from the cones, or a South Dakota air-column blower to remove debris, wings, and hollow seeds. Meanwhile, the approved methods included (1) thumping opened cones vigorously on the side of a box, bottom-screened with a mesh allowing gobs of fresh cone pitch smaller than the seeds to exit; (2) impaling larger gobs of fresh pitch on a pencil point; (3) hand chafing to remove wings (hopefully without many sticky lumps of pitch, seed, and wings); and (4) transferring the resulting mess of seed, wings, dry pitch lumps, broken needles, bud scales, and so forth, to a clean, fine-screened winnowing box. Then, firmly holding the winnowing box over a bedsheet, we made a series of about 20, ever-quickening deep knee bends. The first 5 or so moderately fast deep knee bends "floated" off the broken wings, larger pieces of needles, and bud scales. Then a meticulously executed and quickened ballet of about 15 deep knee bends at the bottom followed by a quick side shift of the seed winnowing box floated off the hollow seeds. We became expert and undeniably proud of our prowess at this exercise. In fact, all debris was removed, and cutting tests showed over 99 percent of the hollow seed removed. Nevertheless, one evening as we were demonstrating the artistry of the winnowing process to a group of visiting Forest Service brass, one of the brass dismissed the entire demonstration with one remark . . . "Ah, the Egyptians were doing that with cereals 2,000 years ago."

[5]Apparently pine squirrels (locally the Richardson's red squirrel, *Tamiasciurus hudsonicus richardsonii* Bachman) stole bagged cones only when near starvation. For instance, in 1951 a very poor year for female strobilus production, coupled with a late June frost, killed most of the already scarce *P. monticola* and *Pinus contorta* Dougl. (lodgepole pine) strobili on two of our selection areas. It developed that the *P. monticola* strobili protected from frost damage by our already installed pollination bags constituted a large proportion of the cones that would mature in the area in the fall, one year later. Thus, one year later the squirrels there were desperate for food, and they went to work on our cone bags. In one selection area the squirrels cut off and lugged away all the bagged cones we had managed to pollinate. In another area, they demonstrated a remarkable selectivity in their thefts. The squirrels had bitten through the folded necks of the cone bags, cut off at the peduncles all eight cones representing the intraspecies cross (*P. monticola* × *P. monticola*), dropping them down about a foot into the bottoms of the pendulous cone bags. Then they had somehow clung to the swinging bags, chewed small holes in the bottoms of the bags, and fished out the almost mature cones through those very tight-fitting holes. They missed only one of the cones. It was found, along with some seeds from other cones, lying in the bottom of a holed bag, and when extracted contained 206 filled and 1 hollow seed. The eight cones representing the interspecies cross (*P. monticola* × *P. koraiensis*), however, were almost ignored by these squirrels. These cones contained more than 1,000 seeds, but every one was hollow

(Wright 1959; Bingham, and others 1974—both report that repeated attempts to produce this hybrid have resulted in only a few filled seeds of doubtful hybridity).

[6]Five-pound size, cotton flour sacks—the standard blister ruster's nosebag in which he carried his sack lunch tied to his belt at the small of back—were used as cone bags. These flour sacks, apparently constructed from yard goods textile millends, were a never-ending surprise and delight. They were printed in a wide variety of brightly colored and imaginatively patterned checks, calicos, and floral designs for use by thrifty home seamstresses. A tall white pine bagged with these flour sacks indeed was a sight; surprisingly the wildly colored bags did not deter the squirrels or cone insects from attacking unbagged cones.

[7]This soil plug cutter had been well-designed by John Breakey and worked beautifully in the loam soils at Fernwood and Elk Creek, Idaho, plots; but it was another matter when we transplanted into the compact, rocky-gravelly soils of the Randolph Creek, Mont., plot. As shown in figure 6, the plug cutters had stout, T-bar pipe handles and foot "rests," the latter on which the operator jumped—once in loam soils and repeatedly in gravelly-rocky soils—to drive the cutter down to the desired depth, attained when the footrests contacted the ground surface. At Randolph Creek, we transplanters all developed a chronic soreness of the feet soon known as "plug-cutter's instep." And later, as we crawled for days on end examining small seedlings for rust, this same gravelly-rocky soil was associated with another malady known as "progeny tester's knee."

[8]This Steering Committee had as members most of the then very few northwestern and California forest geneticists, a few local agronomic crop breeders, forestry faculty silviculturists from local universities, a few blister rust pathology and white pine silviculture researchers, Blister Rust Control, FS Division of Timber Management administration, and Northern Rocky Mountain Forest and Range Experiment Station personnel. It was the forerunner of the Northwest Forest Genetics Association.

[9]In late August 1957, Tony Squillace and the author, along with assistants Doyle Romans, George Blake, and Bob Hill, had just concluded the usual 2-week session inspecting for rust and resistance reactions, measuring tree heights, and weeding and otherwise maintaining progeny tests on the Fernwood, Idaho, field outplanting plot. At the end of the day the five of us sat together on a satisfyingly large and soft pile of weeds just cultivated from the progeny test rows, and we talked about where we might go from there. It had become apparent to us that blister rust resistance in Inland Empire western white pine was under strong genetic control and that resistant planting stocks should be attainable. As I remember it, it was Tony who first voiced the obvious question, "Well, now that we've got resistance, what are we going to do about it?"

We decided we could progress most rapidly toward a practical level of resistance if we could now "take the program out of the woods" into some nearby, long-growing-season area. Our erstwhile Californian cooperator, Jack Duffield, had recently transferred from the Forest Service to lead a new genetics program at Nisqually, Wash., for the Industrial Forestry Association. There, Duffield had just completed planning and constructing a $17,250 facility that included an office lab building, greenhouse, headhouse, lathhouse, and a garage-storage building. This research facility seemed to meet our small needs as well, and if we could only find a couple of suitable and free acres of land to put it on, along with a small nursery, plus 40 nearby acres for a breeding arboretum, we would be all set. Then the impromptu bull-session dissolved as we hurried from the weed pile to the carryall vehicle and back to Clarkia in time for dinner.

At the dinner table, Homer Hartman, supervisor of the local blister rust control force, told us that some brass from the Forest Service Washington Office and the Regional Office in Missoula, Mont., were inspecting blister rust control work to the north around Priest Lake, Idaho. Unbelievably, they were running a day ahead of schedule, and wanted to spend their extra day reviewing the new resistance research. So it was that arriving at Clarkia that evening were Assistant Chief of the Forest Service for State and Private Forestry, Bill Swingler; his Deputy Assistant Chief for Disease Control, Connie Wessela; Region 1 Regional Forester, Pete Hansen; and his Assistant Regional Forester for the Division of Blister Rust Control, Swanny Swanson.

The next day we ferried the inspection party, in two carryalls, first to nearby Crystal Creek to observe rust-free selections in the wild—their healthy branches often interlaced with multi-cankered branches of nearby, rust-susceptible trees. We saved the *piece de resistance* (pun unintended)—the Fernwood Progeny Test Plot—for the last. Luckily the four successive progeny tests, then 1 to 4 years after being artificially inoculated, were in one of their more striking phases. On the one hand, in the youngest test were heavily needle-spotted row-plots of trees from susceptible progenies alongside lightly spotted or almost unspotted row-plots of trees from resistant progenies. On the other hand, in the oldest test were clearcut, red-foliaged row-plots of dead and dying trees of control or other susceptible progenies, often alongside the surviving, green-foliaged row-plots of resistant progenies. After acquainting the inspectors with the 10-seedling row-plot, randomized block design, we suggested they (1) recall the four rust-free parent selections (17, 19, 22, and 58) that they had just finished viewing in Crystal Creek; (2) search out on the row-plot stakes those progeny row-plots having those four selections as one or especially both parents; and (3) inspect the trees in those row-plots closely and carefully, comparing them to trees in adjacent row-plots.

The inspectors dispersed into the progeny tests and we researchers resumed our soft roost on the weed pile; it was highly satisfying to see them emulating our behavior of the last 2 weeks—on their hands and knees, crawling in the narrow aisle between row-plots and peering closely, heads down and tails up, at the small, mostly 6- to 18-inch (15 to 45-cm) trees. After many head-to-head discussions across progeny rows, and after numerous questions to us researchers, the inspectors joined us on the weed pile.

Assistant Chief Swingler probably crawled down the most rows and peered at the most seedlings; at least he was last to return to the weed pile. Then, having just barely taken a companionable seat on the pile, he thoroughly startled Tony and me by paraphrasing Tony's question of the previous late afternoon: "Well, it looks like you're onto something here. What do you think you should be doing about it?" I flicked Tony a glance, and while he seemed to be preoccupied, he did manage

a small nod. Then with a hardly perceptible pause, I launched into an extrapolated version of the previous day's discussion, hoping to leave the impression that the lists of new research jobs and people, research facilities, and operating funds were the result of long and deliberate discussion. Nobody had to write Bill Swingler, or for that matter the other three inspectors, a letter. Bill responded, "Okay, it sounds reasonable. Now you find some free lands for your proposed research installation and I'll see if I can find the money."

About 2 weeks later Connie Wessela was on the scratchy, rural telephone line, calling from Washington, D.C., and saying, "Bill Swingler has raised the $17,250 for your building. Now you get going on the land!" Then Tony and I spent a frantic 2 weeks searching the warmer, western edges of the northern Idaho white pine country for 40 or so flat and potentially free acres of National Forest, BLM, or even State land. We were even beginning to consider purchasing private lands. Then Dean Ernie Wohletz of the College of Forestry, University of Idaho, relayed the welcome news that, because the University Agronomy Department was moving off some campus lands, 40 acres of fertile Palouse farmlands would become available on the University Farm about a mile west of the main campus against the Washington State line. The dean also said a couple more acres would be available from the Forest Nursery (nearer the main campus, along Moscow, Idaho's Main Street) for an office-laboratory-greenhouse-nursery facility.

Within another 2 weeks University-Forest Service cooperative agreements were signed covering free use of these university lands for the establishment of a "Northern Idaho Forest Genetics Center." Almost immediately we began balling, potting, transporting, and transplanting truckloads of 1- to 2-ft (30- to 60-cm) tall, rust-resistant, F_1 seedlings (about 1,000 trees) from the three Idaho and Montana outplanting plots onto the new University Farm Breeding Arboretum (fig.15A and B). Currently, the Genetics Center is an annex off the larger Intermountain Forest and Range Experiment Station's Moscow Forestry Sciences Laboratory that was constructed in 1963 (fig. 16).

[10]From results of the 1952 progeny test (tables 7 to 9), we earlier workers had concluded that major genes could not be associated with the resistance we had measured and, instead, that resistance was quantitatively inherited. Thus, the conclusions drawn from the early test were opposite those from Ray and Geral's tests, and there was no explanation we could suggest to explain the discrepancy. Fur flew when the two scientists first approached me with partial results from a single progeny test. The problems of the cereal rust resistance breeders with collapsing major gene resistance had been pounded into me until dominant and recessive resistance genes had become almost anathema to me—especially when they cropped up in someone else's data. Ray and Geral emerged from the first few stormy sessions with me somewhat battered, but unbowed. Then, styling themselves as the "young Turks," and trailing a somewhat less than real aroma as downtrodden young scientists, they produced more evidence of major-gene-control of the spots-only syndrome from other progeny tests and from self-pollinated progenies. Slowly they coaxed or prodded me into their corral until I reached the point where I was almost enthusiastic about their major-gene hypothesis. Now I have to admit that their persistence and open criticism had become one of my strongest assets as a research administrator.

(A) (B)

Figure 15.—The Moscow, Idaho, Breeding Arboretum: Photo A.—at the time of establishment in 1957. Photo B—20 years later in 1976.

Figure 16.—The Northern Idaho Forest Genetics Center, 1958; now an annex to the Intermountain Station's Moscow, Idaho, Forestry Sciences Laboratory.

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Methods, results, and conclusions are reviewed for a 25-year, USDA Forest Service first-phase Research and Development program. The program goal was to attain useful blister rust resistance in western white pine. One study result is that the Service will be mass-producing second-generation, rust resistant white pines for the Inland Empire. About two-thirds of these seedlings are expected to withstand severe exposure to local races of blister rust. The resistance is based on a number of differential and uniform types. Second-phase work, aimed for fruition before the year 2000, is outlined.

KEYWORDS: host:parasite systems, vertical resistance, geographic variation, altitudinal variation, *Cronartium ribicola, Pinus monticola,* disease resistance, horizontal resistance, tree resistance, seed orchard technology

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